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CLINICAL DEPRESSIONS*

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THE purpose of this paper is to discuss a few points in connection with early or incompletely developed depressive illness. Depressions, like neuroses, impose themselves on the physician and require to be differentiated by him from the neuroses and from primary physical disorders. They are more common than has been supposed but are often difficult of diagnosis, because the emotional change is subtle and because the patient seldom complains spontaneously of being depressed, and seldom presents the objective signs of melancholy.

These incipient depressive disorders are frequently in reality the larval or incomplete forms of either the involutional depression or the manic depressive depression, but they bear little resemblance to the fully developed clinical picture of these two affective psychoses. Moreover, they may wax and wane for years without ever becoming overt mental illnesses in which the emotional change is dominant.

It is a striking fact that scarcely ever does the depressed patient who comes to the physician complain spontaneously of feeling despondent or melancholy. And only the most profoundly depressed patients show objective despondency or retardation. There is no symptom complex pathognomonic of depression. In these incipient cases, the symptoms emphasized by the patient are usually referable to his body, but secondary symptoms suggesting his emotional disturbances are almost always present if sought for. The common symptoms can be divided conveniently into two groups: somatic and psychic.

Somatic: Fatigue, lassitude, dyspepsia, palpitation, dizziness, headache, (often migrainous), flushes, paræsthesiæ, muscle and joint pains, dysmenorrhœa, impotence.

Psychic: Insomnia, tenseness, poor concentration and memory, loss of interest, loss of libido, irritability, panicky feelings, crying spells, guilt feelings.

The patient almost invariably interprets his symptoms as indications of physical disease and even when on direct questioning he admits subjective despondency he often attributes his mood to his body ailments. The reasons for this are several: (1) When an individual feels ill he turns first to his body for explanation. There is a prejudice amongst the laity just as there is amongst the profession, against anything that might be thought of as "mental" disease. Moreover, most people have been indoctrinated so thoroughly into the horrors of cancer, tuberculosis, hypertension and syphilis that they walk in fear of the time when their bodies will let them down. (2) Actual physiological dysfunctions productive of symptoms can be and are produced by emotional disturbances. (3) The emotional depression is characterized psychopathologically by conflict based on feelings of guilt, resolution of which requires the body to be "blamed". The result of all this is distortion of the history as it is presented to the physician. The physical complaints tend to be emphasized and the basic emotional disorder concealed.

The incidence of depressive illness is high amongst neuropsychiatric cases. Kirchof[1] found one-fifth of 1,955 such cases to be depressions. Tredgold[2] found one-quarter of patients in an army mental hospital to be suffering from some form of depression. But this method of analysis gives no indication of the incidence of non-psychotic cases which may appear in the office of the physician. Hohmann[3] emphasized that the depression is frequently masked and therefore missed by the physician in his diligent search for physical disease or his ready acceptance of neurotic motivation.

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He goes so far as to conclude that "the overwhelming number of neurotic states are in reality mild or severe depressions". Ziegler[4] analyzed the records of 111 ambulant patients of all ages who had at first come to the physician or surgeon for the relief of seemingly bodily ailments and who were subsequently observed by the psychiatrist and treated for depression as their chief symptom.

My own experience has convinced me of the necessity of carefully assessing the emotional state of every patient, even those who present some physical basis for their presenting symptoms. As an example:

Mrs. S.: This patient was a woman of 48 whose primary complaints were epigastric pain and tenderness, tiredness, insomnia and nervousness. Physical examination showed that she had a small tender epigastric hernia. It was noted that she was depressed and somewhat agitated. She was fourteen pounds below her usual weight. Symptoms had been present over eighteen months. The epigastric hernia was discussed in consultation with a surgical colleague and it was decided to treat the depression before anything else was done. She was given six electro shock treatments which terminated the depression. She has since been followed for five months. The epigastric hernia is still present but she does not complain of it. Depression and agitation have disappeared. Sleep is normal.

Another patient, Mrs. M., aged 64, complained of severe gaseous dyspepsia, nervousness, irritability, fatigability and insomnia. She was found to have a small hiatus hernia. An internist colleague felt that her complaints were out of proportion to the size of the hernia. She was depressed and agitated. All her symptoms, including dyspepsia cleared up following electro shock therapy. She has been followed for three months.

Although physical disease may be associated with or indeed may precipitate a depressive illness, treatment of a supposed physical ailment in the presence of an unrecognized depression explains many a therapeutic failure. It is dangerous to ascribe fatigue, insomnia, dyspepsia and palpitation—common symptoms in the physician's consulting room—to anæmia, concealed infection, avitaminosis or thyroid disease without enquiry into the emotional state of the patient.

The most difficult distinction, however, is from neurosis. These patients almost always impress one at first as being neurotic, and only by keeping the possibility of depression in mind and by making inquiries relative to the mood of the patient can masked depression be detected or excluded.

Depression and neurosis may be associated in various ways. For instance, morbid depression may activate neurotic behaviour. The reason for this is difficult to comprehend unless one looks upon neurotic behaviour as a certain type of morbid reaction occurring in susceptible or "predisposed" individuals in response to undue stress or strain. Such strain may occur in the form of physical disease, emotional shock, chronic environmental frustration or the onset of a depression. In other words, any force or circumstance noxious to the personality may result in neurotic behaviour. Thus the history of the depressed patient may bristle with accounts of syncopal attacks, hysterical outbursts, acute panic states, obsessive compulsive features and hypochondriacal attitudes. This is why most clinical depressions are mistaken for psychoneurotic reactions. I have at present a depressed patient of cyclothymic personality who, in her current depression, has developed a breast cancer phobia with mastalgia. The essential feature is the depression not the phobia or the tender breast. These will clear up when her depression is treated.

On the other hand, neurotic individuals are not exempt from depressive illness, and the two conditions may exist simultaneously and independently in the same person. In such cases one may be left, after treatment of the depression, with a persistently neurotic, maladjusted individual. The neurotic symptoms do not seem to clear up in this situation as they do when they occur as a side effect of the depression. The differential point, whether the neurosis or the depression is the basic disorder, is so difficult sometimes that Solomon[5] has suggested that in some cases it is justifiable to try the effect of electro shock therapy as a diagnostic measure.

Many cases of depression bear a striking similarity to neurocirculatory asthenia. Indeed the symptom complex of effort syndrome not infrequently constitutes the guise in which a depressive illness manifests itself. Earlier clinical studies of effort syndrome (Lewis,[9] Parkinson,[10] and Fraser[11]) are remarkably free from any consideration of the emotional life of the patient, and in this indicate the insistence of clinical medicine on the strict physiogenesis of an illness that is to be considered authentic. Lewis[6] remarked that his patients were "of highly strung, nervous temperament; an unusual number are sensitive or querulous, others are apathetic or depressed". But he simply attached no clinical importance to these observations. Such data were to him beyond the realm of medical science.

In Paul Woods'[7] series, which was studied psychiatrically by Aubrey Lewis[8] the diagnosis of a psychiatric disorder was made in 55%. Most of these were neuroses but 12% of all cases were in reality depressions. As Lewis points out, there is a wide gap between a symptom and a diagnosis.

Two cases illustrate depressions which exhibited all the symptoms and signs of effort syndrome.

Mr. T., aged 37, weight 160 lb., height 5 ft. 11½ in. first reported for examination in September, 1943. His symptoms were gas on stomach aggravated by all foods and occasionally relieved by milk of magnesia. There was much epigastric distress. When this was severe, pain radiated through to the back. He also complained of dizziness, fatigue, tachycardia, palpitation, precordial pain, loss of weight of fifteen pounds in three years and constipation. His history indicated that in 1940 he had consulted his physician because of palpitation and weakness. He was reassured by the doctor on the first examination but at a second examination he was told he had some trouble with his heart. He then went to an internist who told him he had no heart disease. As he had many pains he consulted more doctors and went to nineteen in all between 1940 and 1946. No abnormalities were found on physical examination. X-rays of the stomach and duodenum, gall bladder and colon were normal. Two gastric analyses showed normal acid. Urinalyses and blood counts were normal and a basal metabolism gave a reading of minus 1%. He was treated by the administration of a smooth diet, cremalin, phenobarbital, siblin, beminal and charcoal.

A surgical colleague referred him to me in June, 1946 with the notation that "this patient is extremely nervous. He complains of a choking sensation and is afraid that he will choke to death some day. . . ." He was noted to be depressed and agitated. Main complaints were gaseous dyspepsia, constipation, palpitation, precordial pain and tachycardia, lassitude and fatigue. Previous history was not significant until 1940. He had come to Canada from Middle Europe as a child; he was married and had five children ranging in age from one to thirteen years. He wept several times during examination. A diagnosis of depression was made and he was given eight electro shock treatments between June 24 and July 11. These relieved his fatigue, palpitation, tachycardia and dyspepsia and he returned to his normal, vigorous state of mild hypomania.

Mrs. P.: This patient was a 35-year old farmer's wife. Her early life had been free from illness. She had married at 22 and was the mother of two children ages 9 and 7. She was admitted to hospital April 10, 1946 complaining of what she called heart trouble. This consisted of palpitation, weakness, precordial pain and a persistent idea that her heart was weak. These symptoms had been present for six years. She had been constantly in bed since the autumn of 1945 and intermittently in bed for prolonged periods before that because of palpitation, weakness and dyspnœa.

When put to bed in hospital she refused to lie down, saying she had not done so for many months as she became dyspnœic when recumbent. Neither could she be persuaded to get out of bed for any purpose whatever. Pulse varied between 80 and 124; blood pressure 128/82. The heart was normal on physical examination. The electrocardiogram showed sinus tachycardia.

She was pale but a blood count showed no anæmia or other blood disorder. The day after admission she became febrile and the third day chest x-ray showed consolidation in the right upper lobe. She was ill with pneumonia for about a month. She never once lay down during this severe illness but was quite co-operative otherwise. She still complained of her original symptoms. Her mood was one of fixed melancholy. She was convinced her heart was not strong enough to carry on and she talked about this incessantly and about nothing else. She remained pale and haggard looking and slept poorly —always upright. She was discharged from hospital on June 3.

Her condition was exactly the same on her return to hospital on July 31. She had remained constantly in bed while at home. Tachycardia was constant and she slept so lightly that a sleeping pulse was not obtained. Electro shock treatment was begun August 14, and she was treated three times weekly. After the fourth treatment she lay down on her bed spontaneously. After the fifth, she slept with only one pillow. After twelve treatments she was up and about helping on the ward. She was spontaneous, good humoured, interested. She felt vigorous and had lost her fear of heart weakness. Palpitations did not bother her. Precordial pain was gone. Her pulse was still fast but she had stopped feeling for it. She was discharged symptom-free on September 14.

The most common form of depressive illness, however, is seen in the female during the involutional period. Such patients present the familiar history of vasomotor and digestive disturbances, associated with a changed menstrual cycle, insomnia, fatigability and tension. Such cases are usually treated by the administration of œstrogenic substances, sedatives and reassurance. Without doubt this treatment is efficacious in the mild cases. But it is entirely inadequate in the more severe cases complicated by agitation and depression, hysterical outbursts or intractable hypochondria. When these occur treatment of the disordered neurovegetative system must be combined with treatment aimed at relieving the depression. Such states of anxious depression may persist for years, if untreated. They may on the other hand run an intermittent course. It is a common experience to be told by a fifty or sixty year old woman that she suffered a previous nervous illness five or ten years previously for the first time in her life, *i.e.*, at or about the time of the menopause. These are involutional disorders and may develop, often in response to environmental stimuli, at any time from the onset of the menopause to well into senility. The longer they occur after the menopause, the less conspicuous become the vasomotor disturbances. But the agitation and depression, sometimes developing into a frankly psychotic state, remains the same as in those of the younger age group. The manifest querulousness, restlessness, irritability and insomnia are sometimes mistaken for symptoms of cerebral arteriosclerosis, or attributed to irreversible organic senile changes.

SUMMARY

1. Depressive illnesses are common in both psychiatric and general medical practice. A large proportion of emotional disorders seen by the physician are depressions rather than neuroses.

2. The incomplete forms of these disorders are seen by the physician rather than the psychiatrist.

3. Identification of these disorders as causative in the production of symptoms is difficult since they may occur concurrently with independent physical disease and neurosis may disturb physiological function and may activate neurotic reactions.

4. Diagnosis will be made only if a routine appraisal of the emotional state of every patient is included in the medical examination.

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RÉSUMÉ

Les syndrômes dépressifs se rencontrent fréquemment, aussi bien en psychiâtrie qu'en médecine générale. Un grand nombre de manifestations émotives observées par le médecin sont plutôt de simples dépressions que de véritables névroses. Les formes frustes et non délirantes sont plus souvent rencontrées par le praticien de médecine générale que par le psychiâtre. Il est difficile d'attribuer une action définie à la dépression dans le déclenchement des symptômes alléguées par le malade mais on sait que les manifestations névropathiques peuvent influencer défavorablement les fonctions physiologiques de l'organisme. On fera le diagnostic de la dépression simple en observant soigneusement la composante émotive du syndrôme présenté par le malade. JEAN SAUCIER

Most diseases do not influence the electrocardiogram. Intoxication, anoxemia, and inflammatory processes are not distinguishable. Alcoholic excess, narcotics, cerebral hæmorrhage, uræmia, shock, and diabetic coma may produce curves suggesting coronary insufficiency, acute or chronic. Thyroid dysfunction and avitaminosis cause irregular changes which disappear as the patient recovers, unless organic heart disease is present.—L. N. Katz and L. G. Kaplan.

STUTTERING: THE PROBLEM AND THE PHYSICIANS' RESPONSIBILITY

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THERE is today among us an affliction whose victims run into the thousands. Centuries of acquaintance with this affliction and centuries of neglect of its victims seem to have instilled into our minds an almost fatalistic attitude. There are approximately one million stutterers in the United States, one-tenth that number in Canada. A great variety of methods have been used to alleviate its distress, surgery, psychoanalysis, witchcraft, hypnotism, drugs, etc., but none of these has had any effect. The medical profession has largely ignored this problem, and naturally the stutterer has turned elsewhere for help and guidance, very often with disastrous results. Thousands of stutterers every year are victims of "quack" schools who guarantee cures where no cures exist.

At first glance, one is amazed by the variety of theories concerning the etiology and treatment of stuttering. Much of the research that has been carried out has been sterile; many workers in the field of stuttering have spent more time in defending their theories than in conducting investigations to discover more pertinent facts about the abnormality. This seemingly wide variance of opinion is unfortunate in that it leads one to assume that there is very little known about stuttering. Even the layman has not hesitated to add fuel to the already glowing fire of confusion and the stutterer receives benevolent advice from all sides.

However, there is some order in this seeming chaos. Day by day the mist surrounding the stutterer is becoming less dense, due in large part to the very active research that is being conducted at many of the large American Universities. The stutterer has been observed from all sides, careful case histories have been taken, and thorough physical, pathological, biochemical, physiological, and psychological studies have been conducted. Contrary to general opinion, these scientific studies tend to indicate that stuttering is not caused by a physical or psychological abnormality, but that it is a form of learned behaviour that could be produced in any "normal" child. Once learned, it is very

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difficult to "unlearn". Personality studies seem to indicate that most of the personality problems of the stutterer are the result of the many penalties he has experienced because of his defect and do not play a significant rôle in the basic etiology. Any theory concerning the etiology of stuttering must take account of the fact that the stutterer does not always have difficulty with his speech. The "average" stutterer can speak quite fluently when he talks or reads aloud to himself, when he sings, when he reads aloud in unison with other stutterers, etc.

In an interesting book by Dr. Wendell Johnson, entitled "People in Quandaries",[1] the point of view is developed that man is the only creature able to talk himself into difficulties that would otherwise not exist. Various abnormal behaviour patterns, particularly the many forms of anxiety tension, can be attributed in large part to the unscientific manner in which our language is used. Many people, for example, develop marked inferiority complexes merely because they feel frustrated at not being a "success". Upon further inquiry, we find that the goal they have been constantly striving for is defined in terms of a word that has no absolute meaning, and thus they have no way of determining when they have attained it. In the verbal usages of our culture, there tends to be no "in-between", the only alternative to "success" is "failure". Similarly, by the misguided use of our language, we have managed to talk thousands of people into evaluating themselves as stutterers.

When scientific observations are made, we find that there is no exact dividing line separating people into two categories, stutterers and non-stutterers. Any attempt to describe the difference in the overt speech of the "stutterer" and "non-stutterer" is an almost impossible task. We could say that stuttering is speech characterized by a large number of hesitations, repetitions or interjections. However, if our listener, trained in scientific thinking, were to ask "How large is large?", we would find it a difficult question to answer. The exact number of hesitations or repetitions necessary before speech is termed stuttering varies from one individual listener or judge to another; no definite answer is possible. We would hit another snag if we were asked to define "abnormal". The author once took part in a short study in which the members of the audience, all trained in speech pathology, were asked to tabulate the number of times the speaker stuttered during a five-minute talk. The number of blocks in the speech, considered by the individual listeners as abnormal varied, from three to forty-two. If such a variance exists among the experts, one can imagine the confusion reigning among the laymen; and stuttering is practically always "diagnosed" in the first place by laymen, usually parents.

After careful observation of the speech of so-called normal speakers, one is amazed by the number of hesitations, repetitions, the number of "ahs" and "uhs", etc. I knew a college student who had entered the speech clinic convinced that he was a "stutterer". So thoroughly convinced was he that he stuttered, that hours of persuasion to the contrary proved ineffective and it was only after he had observed other "severe stutterers" in the clinic that he was finally able to change the evaluation he had always had of himself.

In a study by Tuthill,[1] the phonographically recorded speech of stutterers and non-stutterers was played before various groups of listeners. Not only was there a wide variance of opinion as to the number of blocks in the stutterers' speech, but also, and this is important, 75% of the audience tabulated stuttering blocks in the speech of the normal speakers. Other studies could be cited, all of which tend to indicate that there is no exact line of demarcation separating stutterers from non-stutterers if we are talking in terms of overt speech, and any difference between the two classes can be attributed merely to personal subjective evaluations. In reality, there are only degrees of fluency and non-fluency; at one end of our continuum would be the very fluent speakers, at the other end the "severe stutterers", and in between would be the vast majority who have in their speech a considerable number of hesitations and repetitions. This in-between area would also contain most of the so-called stutterers.

At the State University of Iowa Speech Clinic one is impressed by the number of anxious parents who bring their young children to the clinic "because they were beginning to stutter", although the children's speech, upon examination is usually found to differ little if at all from the speech of any normal child of the same age. Investigations[2] were made of 46 stuttering children and 46 non-stuttering chil-

dren of like age, sex and intelligence level. Relatively thorough observations and case histories were made of the stuttering children over a mean period of two and one-half years. It was found that:

1. All the children in this study had talked without stuttering for from six months to several years before the onset of stuttering.

2. Practically every case of stuttering was originally diagnosed as such, not by a speech expert, but by a layman, usually one or both of the child's parents, the relatives or school teacher.

3. What these laymen had diagnosed as stuttering was by and large, indistinguishable from the hesitations and repetitions known to be characteristic of the normal speech of children.

WHAT IS NORMAL SPEECH?

One obvious fact, but one that is so often ignored, is that before we can diagnose speech as abnormal, we must have a full understanding of the range of normal speech. Investigations[3] were made on the fluency of normal children between the ages of two and six years at the Iowa Child Welfare Research Station and it was found that from 15 to 25% of their words figure in some kind of repetition. The initial sound or syllable of the word is repeated, the whole word is repeated, or the word is part of a repeated phrase. In addition there were frequent hesitations.

The findings can be summarized in another and possibly more meaningful way by saying that the average normal child was found to repeat (a syllable, word, or phrase) forty-five times per thousand spoken words. These repetitions and hesitations are not accompanied by any emotional disturbances. The child seems to be groping for words, "talking over his head", trying to speak while others are talking or not paying attention to him, trying to speak while relatively excited or hurried, etc. Non-fluencies in his speech are the normal result. Since most of the child's movements are unsteady, it should not be surprising that he fails to master immediately the complicated articulatory movements required in speech, especially when we consider that we use structures in speaking that were originally intended for such innate functions as sucking, chewing, swallowing, etc., and also that complex psychological processes are involved.

DIAGNOSIS OF STUTTERING

One is struck by the fact that the so-called stuttering children had normal speech up until the time some one, usually the parents, decided they were starting to stutter. Johnson has coined the word "diagnosogenic". Stuttering is a diagnosogenic disorder in the sense that the diagnosis of stuttering is one of the most important causes of stuttering. This semantic approach to stuttering has partially explained a great many of the unsolved "mysteries" surrounding this disorder. Once the label has been applied, the attitude of the parent toward the child is markedly altered. The parent becomes distressed, and very soon the child begins to change his own evaluation of himself and of his speech. The more anxious the parents become, the more they prevail upon the child to "talk slowly", "start over", "to think before he speaks", "to take a deep breath" and to employ numerous other devices which they assume will help his speech. This constant nagging only serves to convince the child that his speech is not acceptable to the parents. He tries and labours to speak in a manner pleasing to his parents without these normal interruptions and, alas, he is "all thumbs". The more the child tries, the more hesitant is his speech and then the more prompting he receives from his parents, and soon a vicious circle is set in motion. Once that fatal diagnosis has been made, the fire has been ignited and soon the fire spreads. Short, effortless prolongations and frequent repetitions so characteristic of the speech of any child are transformed into the exaggerated hyper-tense emotional speech demonstrated in the adult stutterer.

The semantic environment* of the child plays a very important rôle in determining whether he is ultimately to become a stutterer or not. David Livingstone tells us in all his years of travel in Africa, he did not find a native who stuttered. John Snidecor, in studying the North America Indians (Bannock and Shoshone tribes in Idaho) living under conditions comparatively free from the white man's influence found that not only did they have no stutterers,

* Semantic environment could be roughly defined as the world of attitudes, ideas, opinions, beliefs, parental policies, etc., in which we live and which to a large extent determines our behaviour patterns. In this world of beliefs little attention is paid to the scientific method as a basic orientation in life. In short, it is that part of our environment which is of the least importance to a dog or an oyster.

but they did not even have a word for stuttering. The standards of speech which the Indian child had to reach were so low that every child had satisfactory speech and freedom from anxiety tensions with respect to speech.

INHERITANCE OF STUTTERING

The inheritance of stuttering has occupied a debatable rôle in the theories concerning the onset of stuttering. Some studies have shown that stutterers apparently have slightly more stuttering ancestors than non-stutterers. Any attempt to interpret this difference must consider the fact that stuttering-conscious parents or relatives maintain a continual vigilance to prevent stuttering from occurring once again in their family. For this reason they pay particular attention to the child's speech and tend, therefore, to create the sort of semantic environment in which stuttering is more likely to develop.

TREATMENT

Not only is the overt speech of the young stutterer different from that of the adult stutterer but also the method of therapy is vastly different. In treating the young stutterer, paradoxically, we confine our treatment for the most part to the parents and others who might be influencing the child. The original diagnosis is challenged and attempts are made to reverse the evaluations that have been made of the child's speech. The parents often maintain too high standards for the child not only in speech, but in other forms of behaviour as well. They are urged to be a little less critical, a little more tolerant, responsive, warm, and affectionate. In general, we try to change the semantic environment of the child. If the child's feeling of speech inferiority has not become too firmly fixed, normal speech will usually be the result of this reversal of parental evaluation and policy.

In the older stutterer, we are faced with a problem that is more difficult and complex. The individual has interiorized the evaluations and beliefs of his parents and now regards himself as a "stutterer". Non-fluencies are evaluated as something to be avoided at all cost and most of the stutterer's reactions can be attributed to this desire of avoiding non-fluencies. Normal speakers stumble along unemotionally in their speech, while the stutterer makes a great show of fear and effort whenever he has a hesitation in his speech. He constantly strives to hide the fact that he is a "stutterer", he substitutes "easy" words for "hard" words; he avoids speaking situations; he uses "starters" in his speech to help him over the difficult spots; and in other ways keeps up a running fight with the "stuttering" which he expects and dreads.

In spite of what one reads in newspaper advertisements, the prognosis in treating the adult stutterer is generally poor if we are thinking in terms of ever developing fluent speech. However, if our goal is to develop in the stutterer an objective attitude enabling him to overcome in large part his fear of speaking, our prognosis is much better. Those familiar with stutterers are well aware of the importance of fear in stuttering. The stutterer "stutters" because he is afraid he will stutter, he tries desperately not to stutter and the harder he tries, the more he stutters. It is not so vital that he have fluent speech when he is speaking to himself, or to a dumb animal or to a baby; and since, therefore, the fear of stuttering is usually absent in these situations, speech is ordinarily fluent. However, once the stutterer tries to impress other people with his fluency, failure repeatedly stares him in the face. The stutterer, constantly influenced by his many fluent intervals, ever strives to speak fluently in all situations and rarely succeeds. One can imagine the distress of an expert marksman who misses the target repeatedly during a competition. If it were only possible for the stutterer to stop trying to speak fluently, more fluent speech would soon result. This constant striving for a goal that is unattainable naturally leads to frustration, tension and demoralization.

In the early stages of therapy, the stutterer is asked to make an objective study of his speech, to determine what he actually does when he stutters. His speech is recorded and he listens to it, making deliberate unemotional observations of the pattern of his speech. For the same purpose, he is asked to observe himself as he stutters before a mirror. Attempts are made to challenge his idea that every non-fluency is a stuttering block and that only stutterers have non-fluencies in their speech. He is taught the importance of "fear" as a factor in stuttering. In order to overcome this fear, he is told to do the thing he is afraid to do. Voluntary stuttering is used by the stutterer in a variety of situations that were formerly associated with

anxiety and fear. "Since you are going to stutter anyway, you might as well do it voluntarily" is a philosophy that is emphasized. The old struggle to conceal the stuttering is discouraged and the stutterer is taught to welcome situations giving him the opportunity to demonstrate his stuttering.

Since most stutterers have tried to speak without stuttering for many years and have never succeeded, they are justifiably dubious about ever helping themselves. Lack of motivation is an important obstacle that must be overcome before any treatment can be successful. Individual and group forms of therapy are both used and situational work plays a very important rôle. Throughout therapy, the ultimate goal is not fluent speech but speech that will contain non-fluencies unaccompanied by any emotional disturbance. The stutterer learns to accept the fact that he is a stutterer and, in so doing, achieves automatically a noticeable improvement in his speech as a by-product.

Many speech correctionists attempt to convince the stutterer that he should be able to speak without stuttering. Artificial methods of speaking are used such as arm swinging, "breathy" voice, speaking while relaxed, or with a voice of different pitch, quality or rhythm. The resulting speech usually gives a temporary improvement, but usually these distraction techniques wear out and the stutterer is left far behind his original starting point. Any such method confined merely to overt speech behaviour tends to ignore the underlying psychological factors operating in stuttering. The results are often startling and rapid and the distraction techniques are sometimes easy for the stutterer to use, and thus are the favourites of many of our private speech clinics where quick results are essential for the clinic from a financial point of view. It is safe to say however that such distraction devices are seldom, if ever, used in the more reputable speech clinics.

The scientific study of stuttering is still in its infancy, and of course there are still a number of unsolved problems. A small percentage of stuttering is due to organic causes but in these cases, the symptoms tend to be somewhat different and are easily recognizable as due to some obvious disease. Organic stuttering shows little of the situational variance shown in the "learned" stuttering. Military psychiatrists have attributed some stuttering in the army to a form of "hysteria", but this type of case has not been scientifically investigated to any great extent and no systematic body of knowledge regarding so-called "psychoneurotic stuttering" is available. Reports are legion of stutterers outgrowing their handicap. This phenomenon has been greatly overestimated in the past and is partially responsible for the indifferent attitude assumed by the medical profession. One wonders what the original pattern of the speech of these individuals was and what they actually did to overcome their handicap. In the light of what has been said in this article, many of these former stutterers might not have had speech of the type usually considered as stuttering by speech pathologists. This conclusion is substantiated by the difficulty one encounters in trying to determine what this "growing out of" process really involves. Case histories of former stutterers are notably weak and unreliable and little relevant information has been obtained or published. Some cases reported as having overcome this disorder undoubtedly stuttered, at least to some extent.

The importance of experimental learning psychology is playing a major part in current psychological studies of stuttering and promises some revealing information. In spite of the relative paucity of knowledge concerning stuttering, we are not justified in assuming a "trial and error" method in our therapy. To ignore the stutterer completely is to do a large number of individuals in our society a great injustice. Dr. Fletcher in his book "The Problem of Stuttering" has recognized the fact that training in medicine, *per se*, does not qualify a person to deal with stuttering. Indeed, even the diagnostic and therapeutic principles established by psychiatry in dealing with nervous and mental disorders of the usual sort have so far yielded very unsatisfactory results when applied to stuttering. While training in medicine, and especially in psychiatry, has a value that needs no defence as a preparation for the understanding and treatment of this disorder, it yet remains true that, given such training, one is not necessarily equipped especially to deal with it. The same can be said concerning training in psychology; the average psychologist is about as poorly prepared to deal with stuttering as the average doctor.

This amounts to saying that we must recognize the need for specialization in regard to

this disorder. Though stuttering bears in the main certain marks that are characteristic of other types of disorders, the attempts to deal with it, using concepts derived from dealing with these other essentially different conditions has been responsible, no doubt, for much of our present inefficiency in coping with it.

Prophylactically, the family doctor has a very important rôle to play. It is to him that the anxious parent first brings the stuttering child, and the need for careful advice is essential at this all-important first meeting if we are to prevent the development of a future adult stutterer. Medical schools have neglected this problem in the past and graduating doctors are frequently completely ignorant of this widespread disorder. Case history after case history reports family doctors telling the parents that their child will outgrow it or have been telling the child to speak slower; other cases have been given medicine with the hope that it might help to "slow the child down".

When the need for special therapy is indicated, the doctor should take every precaution to see to it that the stutterer is placed in the hands of a competent speech pathologist. Schools for stuttering advertising cures are to be avoided. So many charlatans have plied their trade in the field of speech correction, due mainly to our own neglect, that the need for reputable clinics has not become apparent. In the United States, more and more universities are opening speech clinics where clinicians are being trained and stutterers being treated. Probably the most important development in this connection has been the growth of the American Speech Correction Association. This organization carries on a vigorous campaign promoting research and guarding against unethical practice.

In Canada, we have recognized the need for supplying aid to those suffering from other handicaps but the stutterer has sometimes been peculiarly neglected. If we can use the averages obtained from American surveys, there are approximately 120,000 stutterers in Canada, or approximately ten times the number of deaf and blind combined. With the growing interest in public health, it is to be hoped that those in charge of the pursestrings will not forget this large group of our population. A sanatorium providing for the stutterer the opportunity of a few months' concentrated speech retaining would usually show significant results.

We must realize that the traditional method of handling stutterers has been far from adequate and that changes are needed in the future. Stuttering now merits our consideration as a disabling and demoralizing behaviour that can be prevented and treated.

The author is indebted for many of the concepts presented in this article to Dr. Wendell Johnson, Director of the Speech Clinic, State University of Iowa, and editor of the *Journal of Speech Disorders*, published by the American Speech Correction Association.

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HIATUS HERNIÆ*

(A new method of demonstrating hiatus herniæ radiologically)

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HIATUS hernia is a subject receiving increasing attention, and a fair literature has grown up about it. There are many young adults wandering about from one doctor to another being x-rayed and re-x-rayed for their gastric symptoms with negative findings. Some have been labelled neurotic to cover up failure in diagnosis and treatment. On account of this apparent frequency of hiatus herniæ, a technique for its demonstration developed in our hospital merits presentation.

A hiatus hernia is the prolapse of part or of the entire stomach through the œsophageal hiatus into the posterior mediastinum. It is usually non-traumatic and is subdivided by Akerlund[1] into three types. (1) The congenitally short œsophagus with all or part of the stomach in the thoracic cavity. This is not a herniation in the true sense of the word, since the stomach

* Read at the 13th Annual Clinical Convention of the Montreal Medico-Chirurgical Society.

develops primarily in the thorax and never descends below the diaphragm. (2) Herniation of portions of the stomach other than the cardia through the œsophageal hiatus. Here the œsophagus is of normal length and the cardia is in its normal position. (3) Herniation of the gastric cardia and adjacent parts of the stomach through the hiatus. The œsophagus is of normal length.

In our discussion we shall limit ourselves to types (2) and (3) and omit the herniæ associated with the congenitally short œsophagus.

As in herniations elsewhere, these can be subdivided into: (*a*) fixed herniæ; (*b*) sliding herniæ.

It is this latter sliding type of hernia that is mostly seen and our discussion will deal with it primarily.

ETIOLOGY

Hiatus hernia may be due to: (1) A congenital weakness or maldevelopment of the diaphragm leading to an abnormally wide œsophageal hiatus. (2) Senile atrophic changes of the peri-œsophageal tissues of the diaphragm.

Schatzki[2] believes that small hiatus herniæ are actually physiological in late life. The loss of fat and the decreased elasticity of the connective tissue of the diaphragm result in relaxation of the hiatus. (3) Conditions leading to a marked increase in intra-abdominal pressure such as: (*a*) severe trauma to the abdomen; (*b*) pregnancy; (*c*) ascites; (*d*) coughing; (*e*) the wearing of tight corsets; (*f*) mega colon.

(4) Malformation of the thoracic or lumbar spine leading to a kyphosis or scoliosis and consequent shortening of the skeletal structure. This causes apparent lengthening and laxity of the crura of the diaphragms which in turn lead to widening of the œsophageal hiatus and hence to hiatal hernia formation.

INCIDENCE

No reliable statistics are available as to the incidence of hiatus herniæ for the following reasons: The condition was practically unknown before the use of x-rays. At post mortem they are often missed due to their transitory character unless special attention is directed to them and special dissecting methods are used. A large proportion of cases are asymptomatic and never recorded. X-ray examination may fail to reveal the condition in a smaller or larger percentage of cases depending on the technique employed.

In the past, hiatus herniæ have been reported in 1 to 3% of all routine barium series. Knothe[3] found 9% in his routine gastro-intestinal series by a special technique of his own. Fineman[4] reported hiatal herniations in 13% of patients examined by a special technique employed at the Montefiore Hospital in New York.

At the Montreal Jewish General Hospital we found 16 cases in the last 100 consecutive barium series done by a technique I will describe later, an incidence apparently higher than that reported by Fineman or Knothe. Harrington[5] found only thirty cases of hiatus herniæ at the Mayo Clinic from 1900 to 1925. It was only when they developed a radiological technique of their own, that they were able to report 147 cases from 1925 to 1933, a period of eight years.

PREDISPOSING FACTORS

(*a*) *Sex.*—According to Fineman there is no difference between males and nulliparous women. Bokus[6] reports an incidence of 10 females to 1 male while Eisen[7] claims an incidence of 2 females to 1 male.

(*b*) *Age.*—Bokus[6] claims that the majority of hiatus herniæ occur in people over the age of 50, while in Fineman's experience they rarely occur before the age of 30, pregnant women excepted. In a series of 221 cases Fineman reported an average of 49.5 years. This has not entirely been our experience. True enough, we meet with many hiatus herniæ in elderly people, but we have found them often in younger individuals.

HABITUS

Hiatus herniæ are most commonly found in hypersthenic obese individuals, but they are by no means the only group affected. I would like to mention a case of hiatus hernia in a girl of 24, whose complaints were gastro-intestinal and who had x-ray investigations to reveal a "low stomach and fallen kidneys", but whose symptoms would be aggravated by any attempt to correct the latter by abdominal supports etc. The reason was obvious, as any attempt to support the stomach would increase the intra-abdominal pressure and so aggravate or induce the hiatus hernia with its train of symptoms.

CLINICAL SYMPTOMATOLOGY

Patients may be entirely asymptomatic or show a variety of symptoms which are related mostly to meals and certain postures.

(1) *Pain.*—This is usually described as a feeling of constriction, pressure, burning, pinching or pulling sensation in the epigastrium or under the lower half of the sternum, and is usually referred to the back between the shoulder blades into the upper thorax to the left shoulder and arm, almost suggesting angina pectoris. The pain usually comes on after meals and especially at night on retiring after a heavy meal. Any sudden increase of intra-abdominal pressure such as coughing, straining at stool or bending over to tie one's shoe laces will bring it on. This pain is usually relieved on the assumption of the erect posture.

(2) Dysphagia is frequently met with, especially if complicated by an œsophagitis or ulceration. (3) Belching, regurgitation and vomiting are common. (4) Pulmonary symptoms such as cough and dyspnœa may be present only in the larger herniations. (5) *Cardiac symptoms.*—The typical anginal pain radiating to the left shoulder and arm has been mentioned above. Pallor, palpitation, rapid pulse rate, dyspnœa, cyanosis and fainting spells are frequently seen. It is not unusual to find one of these patients collapse on the bathroom floor.

The electrocardiographic changes are very indefinite. Fineman,[4] in his exhibit at the New York Academy of Medicine, in April, 1943, stated that "Electrocardiographic findings in hiatal herniæ may be positive, suggesting myocardial disease or damage". The cause or mechanism of these electrocardiographic changes in patients with otherwise normal hearts merits further investigation.

It is my feeling that more uniform electrocardiographic findings could be obtained if they were recorded under fluoroscopic control at a time when the hernia is brought into view by the procedures to be mentioned below. I feel that when the hernia is in the posterior mediastinum encroaching upon the heart, changes in the electrocardiogram at that time would be more uniform, more definite and hence of more diagnostic significance. These electrocardiographic changes should disappear on assuming the erect posture, much the same as the hernia itself disappears. This field of study deserves further investigation.

(6) Signs of blood loss such as hæmatemesis, melena and anæmia have been observed by Morein[7] in about 21% of cases. The latter may be due to an associated duodenal or gastric ulcer, which was noted in 23% of cases or due to an interference with the normal blood supply in the constricted portion of the herniation leading to erosions, bleeding or to a concomitant œsophagitis.

(7) *Associated diseases.*—There are a number of diseases associated with hiatus herniæ. According to Bokus[6] and Harrington[11] the more common ones are: (*a*) peptic ulcer; (*b*) cholelithiasis, especially in the hypersthenic obese individuals in whom hiatus herniæ are frequent; (*c*) herniations elsewhere in the body—this being indirect evidence of a congenital weakness in muscular development; (*d*) diverticulosis of colon or duodenum often associated with redundancy of the colon; (*e*) scoliosis or kyphosis of the thoracic spine.

Of 60 patients operated on by Harrington[11] between 1925 and 1933 at the Mayo Clinic 29 had been treated for cholecystitis and 13 of these had had gall bladder operations; 23 had been treated for "stomach trouble" (18 for ulcer and 5 for hyperacidity); 5 had had a pyloroplasty or gastro-enterostomy; 7 were diagnosed "secondary anæmia"; 4 were diagnosed intestinal obstruction; 10 were diagnosed œsophageal obstruction, of which 5 were thought to be cardiospasm, 2 cancer of the œsophagus and 3 stricture of the œsophagus.

RADIOLOGICAL FEATURES

The routine chest film.—The routine chest film may or may not reveal the presence of a hiatus hernia depending on whether it is of the fixed or sliding type. In the fixed type, the *magenblase* is absent below the left diaphragm, while a collection of air and food is noted above the diaphragm adjacent to the left cardiac border in the regular postero-anterior views. Films of the chest taken in the dorsal decubitus position in deep inspiration may occasionally reveal the presence of a sliding hernia. The upper border of the barium filled hiatus hernia may at times assume a "molar tooth" appearance, which is of diagnostic significance in differentiating the latter from a dilated œsophageal ampulla.

Special procedures.—Friedenwald and Feldman[8] stress rapid, deep breathing and straining during the fluoroscopic examination as a

method of visualizing the hiatus hernia. The patient is examined in the horizontal and Trendelenburg positions, turning him through a 360° arc while under continuous fluoroscopic control. The patient's efforts in turning produce a momentary increase of intra-abdominal pressure which may bring into view momentary hiatus herniæ.

Heavy compression of the abdomen to increase intra-abdominal pressure has been recommended by Knothe and demonstrated by Fineman. A small balloon is placed on the abdomen and inflated. The patient is examined in the right oblique recumbent position during the act of swallowing with his weight on the air-filled bag. Fineman[4] recorded 13% of hiatus herniæ in all his gastric cases when he employed the above technique.

Schatzki[9] distends the colon with air and finds that hiatus herniæ are demonstrated in 70% of patients over 60 years of age by this method. Hurst, however, claims that heavy compression and the distension of the colon with air are manœuvres too strenuous for use in elderly patients and may produce herniations which are never present under ordinary circumstances. Templeton[12] examines the œsophagus during the act of swallowing and makes use of the "Valsalva test" technique to bring the hiatus hernia into view.

The method employed here is incidentally something I did not find recorded in the literature. No balloon, no artificial or extraneous methods of increasing intra-abdominal pressure and no special thicker mixtures of barium or pastes are used. The procedure is simple and easily mastered and has been adopted as a routine in every barium series done regardless of symptomatology.

After completing the examination of the stomach and duodenum in the erect posture, the patient is instructed to take as much of the barium drink as he can and keep it is his mouth. He is not to swallow it until told to do so. The table and patient are lowered into the horizontal or slightly Trendelenburg position. He is positioned in the right posterior semi-oblique thus affording a clear view of the posterior mediastinum.

After proper positioning, the patient is instructed to swallow all the barium in his mouth so as to completely fill the œsophagus. When the latter is entirely filled with barium, the patient is told to take a deep breath, and hold it. A herniation of the cardia, if present, will come into view, along with any diverticula of the œsophagus which could not be seen before this stage of the examination. A dilated œsophageal ampulla will also manifest itself by this procedure. It is to be emphasized that the patient is on his back and not on his abdomen. No increase of intra-abdominal pressure even by his own weight is utilized in demonstrating the hiatus hernia by this method. The "Valsalva test" technique described by Templeton[12] is not employed in order to bring the hiatus hernia into view.

Films are taken on the spot and under fluoroscopic control. The practice of sending the patient from the fluoroscopic to the radiographic room for films may fail to demonstrate the hernia.

To explain the mechanism of the above procedure, I should like to emphasize that the patient lying in the horizontal or slightly Trendelenburg position is told to swallow all the barium in his mouth and only when the entire œsophagus filled with barium is visualized is he instructed to take a deep breath. This deep breath causes contraction of the diaphragms and their crura, thus widening the œsophageal hiatus; it also causes an increase in the intra-abdominal pressure and a decrease in the intra-thoracic pressure. The barium-filled œsophagus acts as a drainage tube ready to obey the laws of gravity and fluid seeking its own level; in other words, acting as a syphon drainage tube to empty the stomach contents into and through the œsophagus. This syphon action aided by the increased intra-abdominal pressure and the diminished intra-thoracic pressure sucks the cardia portion of the stomach through the lax œsophageal hiatus, allowing the latter to manifest itself as a herniation above the diaphragm.

The following cases are presented as illustrations of hiatus hernæ associated with other pathological conditions for which they may often be mistaken.

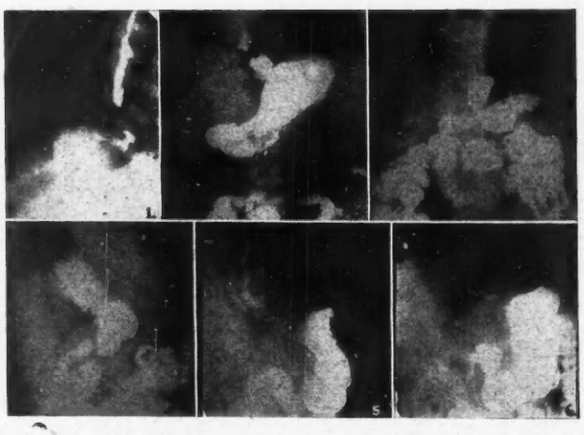

Fig. 1.—Hiatus hernia in a young woman aged 33 associated with scoliosis of the dorsal spine. Note the "molar tooth" appearance of the upper border of the herniated pouch. **Fig. 2.**—Hiatus hernia associated with: (1) kypho-scoliosis of spine; (2) gall stones (to the left of the spine); (3) diverticulum of the œsophagus; (4) enlarged liver and spleen. Patient aged 45. **Fig. 3.**—Hiatus hernia associated with gall stones. Patient aged 67. **Fig. 4.**—Hiatus hernia associated with a penetrating gastric ulcer at level of hiatus. Patient aged 65. **Fig. 5.**—Hiatus hernia associated with an infiltration of the gastric wall within the herniated pouch suggestive of a carcinoma. Patient aged 74. **Fig. 6.**—Same case examined 3 years earlier revealing the presence of a small hiatus hernia associated with an œsophagus of normal length.

CONCLUSION

An attempt has been made to review the most important features of hiatus herniæ, laying particular stress on a method of demonstrating the latter roentgenographically. Their occurrence in apparently young adults is more frequent than usually reported. A coincidence of hiatus herniæ with various other pathological conditions with which they are often associated and for which they have often been mistaken has been mentioned. There are cases of angina pectoris with no evidence of true organic heart disease, in which the attacks are brought on by food and posture rather than by exercise. The question of hiatus hernia as the etiological factor in these cases should always be considered. These conditions merit further investigation by electrocardiographic studies done under fluoroscopic control.

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ŒSOPHAGEAL HIATUS HERNIA*

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THE demonstration of various types and degrees of herniation of abdominal organs through the diaphragm constitutes a notable advance in diagnostic radiology during the past twenty years. This large and fascinating subject of diaphragmatic hernias may be divided into two subdivisions: (1) non-traumatic; (2) post-traumatic hernias.

Hernias through the œsophageal hiatus constitute the largest group within the non-traumatic division. It is to these that we wish to refer. This condition is one of great importance to radiologists since clinical diagnosis on the basis of signs and symptoms is very difficult, whereas radiological demonstration, when proper technique is used, is quite easy. Within relatively recent years, the diagnosis has been made with increasing frequency. Various writers, notably Harrington of the Mayo clinic, have indicated a twenty-fold increase in the diagnosis of diaphragmatic hernias during recent years. The greatest proportion of these were hernias through the œsophageal hiatus. In 1911, Eppinger collected 11 cases of œsophageal hiatus hernia. In 1923, Richards collected 23 cases. Subsequent publications by Akerlund, Morrison, Harrington, Truesdale, and many others, have made the condition well known. One still finds, however, that a fairly large number of patients have been x-rayed once or twice before without the diagnosis being made. The reason for this is, I believe, purely a matter of technique. Harrington, in his series, found that an average of three previous erroneous diagnoses had been made.

The œsophageal hiatus is a relatively small opening, elliptical in shape, and lying slightly to the left of the midline at the level of the tenth dorsal vertebra. It is formed by splitting of the medial fibres of the right crus of the diaphragm. Through it pass the œsophagus, the two vagus nerves, or a plexus of nerves formed from them, and the œsophageal branch of the right gastric artery. When herniation occurs, the hiatus also may contain a portion of the fundus of the stomach, and occasionally also part of the omentum, and rarely also the spleen. The overwhelming percentage of cases involve the stomach only.

CLASSIFICATION

There are two main groups which are distinguished by the position of the œsophagus.

Group I is the para-œsophageal group (see Fig. 1). The œsophagus is of normal length and maintains its usual relationship to the diaphragm. A portion of stomach has protruded up into the posterior mediastinum beside the œsophagus. These hernias are usually relatively small, rarely involving a very large segment of the stomach.

Group II is the "elevated œsophagus" group (see Fig. 2). These were called the gastro-œsophageal type by Akerlund. The œsophagus is displaced upwards into the posterior mediastinum along with the herniated portion of the stomach. The lower œsophagus becomes tortuous to take up the extra length. Such hernias are reducible, as the œsophagus will reach the diaphragm quite easily. This type of hernia

* Read before the tenth mid-winter meeting of the Canadian Association of Radiologists, Quebec City, January 3, 4 and 5, 1947.

may be quite large, including a large part, or all, of the stomach, and even, in some cases, part of the omentum and colon. This type of hernia may develop from the para-œsophageal type. Over a long period of time, the pressure differential between the abdomen and thorax causes more and more of the stomach to herniate. Coincident with this, of course, the clinical picture may show a gradual change which renders the diagnosis more difficult.

Minimal degrees of both of these types are fairly common and have been called pulsion types by Harrington. They are frequently incidental findings, but as they may give rise to marked symptoms, close co-operation of clinician and radiologist is essential for their evaluation.

A third group usually considered in this connection is the congenitally short œsophagus. In these, the œsophagus is straight and would not reach the diaphragm (Fig. 3). The stomach has always been partially intrathoracic. Embryologically, the stomach appears above the developing diaphragm, and moves caudally as the œsophagus elongates. Rarely, the diaphragm may close before the stomach has become completely intra-abdominal. These are said by Harrington to be rare, although some writers have classified a fairly high percentage of their cases in this group, probably because they considered many of the elevated œsophagus types to be congenitally short. It appears, from the experience of others, that the diagnosis of congenitally short œsophagus is seldom justifiable on purely radiologic grounds, except in children, and requires confirmation by œsophagoscopy and operation.

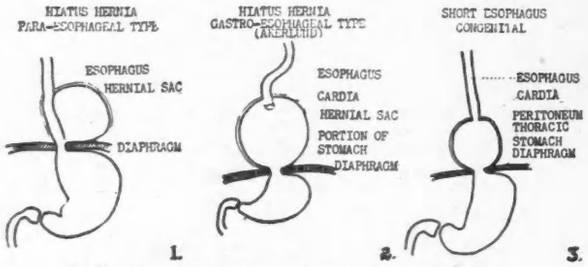

Fig. 1.—Para-œsophageal hiatus hernia.
Fig. 2.—Elevated œsophagus type.
Fig. 3.—Congenitally short œsophagus.

Other conditions around the lower œsophagus which must be distinguished include dilated œsophageal ampulla, cardio-œsophageal relaxation, epiphrenic diverticulum of the œsophagus, cardiospasm, peptic ulcer, cicatricial stricture, and cancer.

ETIOLOGY

The œsophageal hiatus is subjected to considerable strain in persons with high intra-abdominal pressure, particularly in obese persons, and in pregnant females. In the latter group, a high incidence of hernia has been cited by Rigler and Eneboe. One is struck by the fact that the majority of the patients are obese, or at least of very sthenic habitus, often with a more or less transverse disposition of the stomach. Most of them are middle-aged or older. With advancing years, the fibres of the diaphragm, in common with all muscles, lose some of their elasticity and the hiatus tends to stretch somewhat. This makes herniation relatively simple. The hernia passes up into the posterior mediastinum behind the heart. Most writers describe a higher incidence in females, but our series does not conform to this. The twenty-four cases here presented constitute too small a group, however, for any sweeping statistical conclusions.

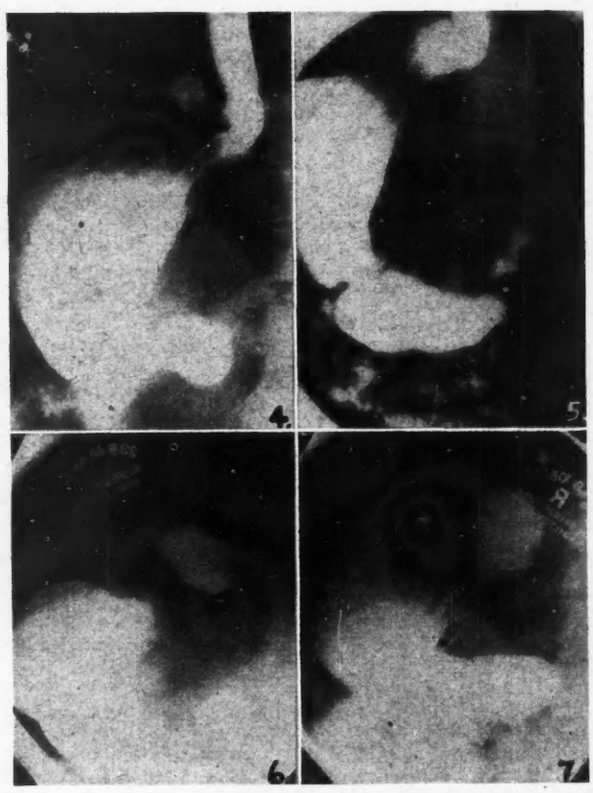

Fig. 4. — Example of para-œsophageal hernia. **Fig. 5.**—Example of para-œsophageal hernia. Patient also had duodenal ulcer. **Fig. 6.**—Elevated œsophagus type in male, aged 50. Pain in epigastrium and substernal region occurring after each *large* meal. Complaints related to *volume* of food, not to *type*. No discomfort after small meals. **Fig. 7.**—Moderately large hernia in male, aged 51, moderately obese. Complaints: nausea, discomfort immediately after meals, with belching, heartburn.

SIGNS AND SYMPTOMS

The patient complains of "heartburn", or an acid sensation behind the lower sternum, often associated with regurgitation of small amounts of very acid fluid into the mouth. The symptoms usually arise after meals, particularly the evening meal, and are often produced or aggravated by lying down. The symptoms follow large meals, and are related to the *volume* rather than the *type* of food. This is an important clinical point. Some of the patients have a history of gastro-intestinal hæmorrhage and others show a definite secondary anæmia of chronic blood loss. Some report gaining relief with alkalis. They have frequently been treated for angina, cholecystitis, peptic ulcer, etc., before the correct diagnosis is made.

DIAGNOSIS

The above noted clinical features are sufficient to bring the condition into the differential diagnosis, but radiologic examination is necessary for confirmation. Unless the proper technique is followed, most of the cases will escape detection. Few of these hernias are visible in the upright position. After the upright portion of the fluoroscopic examination has been completed, we routinely give the patient another drink of barium in the decubitus position by means of a curved drinking tube. A thick barium paste is always kept at hand and the patient is given some of this if the liquid barium does not remain long enough in the lower œsophagus to permit adequate visualization. If any abnormality is noted, spot films are taken. For the benefit of our clinical confrères and others viewing films, we also attempt to demonstrate the hernia on our routine run of stomach films by having the patient swallow a little barium before each exposure. The films then usually show the lower œsophagus, stomach and duodenum all filled with barium. We do not routinely follow any technique for producing gross increases in intra-abdominal pressure as we feel that herniations which only appear under such abnormal circumstances are unlikely to be of clinical significance. Sometimes we have resorted to increasing the pressure to render a hernia more prominent for filming purposes, but this is merely to make more obvious a condition already visualized.

The roentgen diagnostic points include: (*a*) the presence of a sac-like or globular barium mass just above the diaphragm; (*b*) a slight constriction just above this at the site of the junction of œsophagus and stomach; (*c*) a narrowed zone below, where the herniated portion passes through the diaphragm; (*d*) gastric mucosal pattern in the involved segment; (*e*) asymmetry of the œsophagus with relation to the barium mass.

Within a period of about ten months, during which hiatus hernias were routinely looked for, 24 cases were found in 522 examinations of the stomach and œsophagus, an incidence of 4.6%. The literature records incidences ranging from less than 1% to the 73% reported by Schatzki in a group of old patients in whom the intra-abdominal pressure was markedly increased by mechanical means or by dilating the colon with air.

Our cases included 18 males and 6 females. The average age was 48, and ages ranged from 21 to 66. It is interesting to note that the 21 year old patient was very obese. Eighty-three per cent of the patients were over 40 years of age.

TREATMENT

1. *Surgical repair* is the treatment of choice for large hernias, or for those producing severe symptoms, provided the patient is in good physical condition.

2. *Medical treatment* suffices in many cases, and is obligatory in elderly patients in poor physical condition. (*a*) Soft, low-roughage diet. (*b*) Multiple small feedings daily instead of large meals. The stomach must not be overloaded. (*c*) Weight reduction for obese patients. (*d*) Avoid reclining after meals. Elderly patients who require rest should lie down for a while before meals. Some may have to sleep in a semi-reclining position, as many pregnant females with severe "heartburn" are forced to do.

CONCLUSION

1. Œsophageal hiatus hernia is a relatively common cause of gastro-intestinal complaints, which, though not specific, are suggestive.

2. Unless proper technique is followed, many cases will be missed on roentgen examination. All patients having examination of the upper gastro-intestinal tract should be given some barium to drink while in the decubitus position under fluoroscopic observation.

3. Those with large hernias and severe symptoms should be operated upon, if they are in

good general condition. Medical treatment will afford some relief to those unsuitable for surgery.

Since preparation of this paper, an additional twenty-five cases of hiatus hernia have been seen. Three of these have had operative repair.

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RÉSUMÉ

Le hernie de l'hiatus œsophagien, traumatique ou non, est une cause relativement fréquente de troubles gastro-intestinaux. Le diagnostic est difficile par le seule clinique, et le plus souvent, c'est la radiologie qui tranche la question. Trois types de hernies sont décrits. Une technique radiologique rigoureuse doit être suivie si l'on ne veut s'exposer à ne pas mettre convenablement en évidence la hernie soupçonnée. Ces malades doivent boire le baryum en position couchée puis examinés au Fluoroscope. Les malades porteurs de grosse hernie avec symptômes graves doivent être opérés lorsque leur état général est bon. Le traitement médical peut apporter quelque soulagement à ceux pour qui la chirurgie serait aléatoire.

JEAN SAUCIER

All hospital linens which are marked or stamped with an aniline dye should be laundered before use. Cyanosis developed in 3 premature and 32 newborn babies shortly after they had been diapered with cloths freshly stamped with aniline dye and which had not been laundered, report E. P. Scott, M.D., and associates of Louisville, Ky. Three infants with severe diarrhœa and excoriated buttocks became cyanotic after they were bathed with freshly stamped washcloths. One treated intravenously with 2 mgm. of methylene blue in 10 c.c. of normal saline recovered within one hour; the others remained cyanotic from twelve to twenty-four hours. Aniline intoxication may have been a factor in the death, within a week, of 1 of the premature infants.—*J. Pædiat.*, **28:** 713, 1946.

ANKYLOSING (Marie-Strümpell) SPONDYLITIS*

(An Analysis of 100 Cases)

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Toronto, Ont.

EARLY in 1945 a Joint Services Arthritis Centre was established at St. Thomas, Ontario, for the group treatment of rheumatic cases from the Navy, Army and Air Force. Since the termination of the war this Centre has continued to operate in Toronto under the Department of Veterans' Affairs.

During the investigation and treatment of some 850 patients with rheumatic disease many observations of interest have been made. Perhaps the most enlightening has been a study of the cases of ankylosing spondylitis, which revealed not only an unsuspected frequency among those suffering from back pain but, as well, a lack of appreciation of its early diagnostic features.

Few cases were recognized early, when prompt institution of therapy would have been most beneficial. The early diagnoses included most often: low back strain, lumbago, fibrositis, sciatica, osteoarthritis, extruded disc, psychoneurosis, and sacro-iliac arthritis. In this series, an average of 5.7 years elapsed from the time of onset of symptoms to the recognition of the disease.

It would seem of value, therefore, to present a study particularly of the early features which, in retrospect, reveal a somewhat uniform clinical pattern that has not been sufficiently recognized or given the clinical prominence which it deserves. One hundred cases have been reviewed: 66 consecutive cases from the Arthritis Centre, and 34 from the wards of the Toronto General Hospital.

INCIDENCE

Ankylosing spondylitis is by no means a medical curiosity. In a recent study by Boland and Present,[1] 18% of patients admitted to a U.S. Army General Hospital, with chronic back complaints, were found to have this disease. No accurate figures of its frequency in the

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general population are available, but Schmorl[2] found 8 cases among 10,000 spines examined, an incidence of less than 0.1%, while Bachman[3] reported 41 clear cut cases in 2,561 x-ray films of the spine, an incidence of 1.6%. Oppenheimer,[4] in a study of 50 cases, noted an incidence of 3.2% of all vertebral lesions observed roentgenographically between 1934 and 1941. Hare[5] reported 21 cases (1.7%) in 1,179 patients with arthritis seen in a period of two years. Of all new patients seen in an office practice during a five-year period, Herrick and Tyson[6] saw 10 cases (0.2%) of ankylosing spondylitis.

The disease is uncommon in women. In this group there were 94 males and 6 females: a ratio of 16 to 1.

Earlier figures on the incidence of the disease in relation to rheumatoid arthritis have varied from 1 of spondylitis to every 11 to 16 cases of rheumatoid arthritis.[5, 7, 8] At this Arthritis Centre the ratio was 1 case of spondylitis to every 2.8 cases of rheumatoid arthritis, a figure consistent with that reported by Boland and Present[1] who cited an equally high ratio of 1 to 3.

In 73% of the cases in this series the onset of the disease occurred before the age of 30, the average age being 24 years. The youngest age at which symptoms commenced was 12 years, and the oldest 46.

PRECIPITATING FACTORS

The etiology of ankylosing spondylitis is unknown, and in 70% of the cases in this series no precipitating factor was suggested (see Table I). Thirteen patients related the onset of symptoms to a back injury but this might well have been an aggravating factor in a disease already present. The factor of trauma, however, has unusual significance in civilian industrial cases where the amount of compensation in this chronic, disabling disease—apparently precipitated by an accident—is most difficult to assess. Eleven patients gave a history of unusual exposure, chiefly to cold and wet; and 6 gave a preceding history of acute gonococcal urethritis. It is felt by some[1] that in this disease, as in rheumatoid arthritis, gonococcal infection of the genito-urinary tract, like other infections, may sometimes serve as a "trigger mechanism".

TABLE I.
PRECIPITATING FACTORS IN 100 CASES OF ANKYLOSING SPONDYLITIS

No precipitating factor elicited	70%
History of back injury	13%
History of unusual exposure	11%
History of acute urethritis (V.D.G.)	6%

CLINICAL MANIFESTATIONS

In most cases diagnosis was hampered by a slow, insidious onset, but a search for early symptoms revealed a back pain as the first complaint in the majority of cases (82%; see Table II). This pain was variable in character and severity but usually was intermittent at first, with episodes of aching followed by periods of complete relief. Aggravation on coughing or straining was common. Stiffness of the spine following periods of rest, which tended to be relieved by normal activity—jelling—was present in 56% of our cases, suggesting a diagnosis of fibrositis. Pain referred to the region of the gluteal muscles occurred in 35% of cases; to the dorsal spine in 23%; and to the cervical spine in 17%. Twenty-one patients complained of pain with a sciatic distribution but it usually lacked the well defined course of true sciatica, seldom extended below the midthigh and often would alternate from side to side. Associated neurological changes were encountered rarely. Girdle pains in the region of the chest were a common feature, occurring in 25 cases, while peripheral pains in the limbs in regions apart from joints were found in 28 cases. Aggravation of symptoms with weather changes was commonly noted.

TABLE II.
SYMPTOMS IN 100 CASES OF ANKYLOSING SPONDYLITIS

Symptoms at Onset			
Referable to:	Back	82%	
	Peripheral joints	12%	
	Chest	6%	
Later Symptoms			
Low back pain	71%	Chest pain	25%
Stiffness of spine	56%	Peripheral pain	28%
Buttock pain	35%	Weight loss	54%
Dorsal pain	23%	Easy fatigue	36%
Sciatic pain	21%	Weather effect	35%
Cervical pain	17%	Cough aggravation	33%

That ankylosing spondylitis is a systemic disease was adequately supported by the general constitutional symptoms encountered. Weight loss, easy fatigue, and generalized weakness were common features, together with low grade fever, slight leucocytosis, secondary anæmia

and elevation of the erythrocyte sedimentation rate. Such systemic features were seen chiefly in patients with peripheral joint involvement, but advanced degrees of spinal involvement may occur in an apparently healthy individual (see Fig. 1).

The patient with ankylosing spondylitis may present a characteristic, rigid, often anthropoid, gait. Spasm of the erector spinæ muscles with loss of the normal lumbar lordosis and flattening of the lumbar area in flexion (Fig. 2) is an almost constant finding (see Table III). A

TABLE III.
CLINICAL FINDINGS IN 100 CASES OF ANKYLOSING SPONDYLITIS

Abnormal posture: rigid (anthropoid) gait; loss of lumbar lordosis; thoracic stoop; protruded neck; flattened chest.	
Kyphosis	38%
Scoliosis	20%
Reduced spinal mobility:	
Lumbar	96%
Cervical	56%
Reduced chest expansion	70%
Erector spinæ spasm	41%
Spinal tenderness	23%

variable degree of dorsal kyphosis with thoracic stoop and protrusion of the neck may be observed (Fig. 3), while less frequently lateral curvatures of the spine may develop.

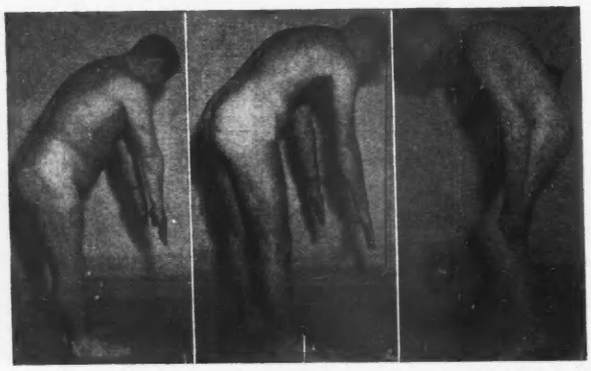

Fig. 1.—Advanced degree of ankylosis in an apparently healthy male. In the upright position, a normal posture was presented; on flexion, a "poker back". (X-ray findings are shown in Fig. 8.) **Fig. 2.**—Ankylosing spondylitis, showing flattening of lumbar area with spinal flexion. **Fig. 3.**—Advanced case of ankylosing spondylitis, the entire spine being completely rigid in the above posture. Note the forward protrusion of the neck and extreme dorsal kyphosis in an untreated case of 30 years' duration. (see x-ray, Fig. 7.)

Spinal mobility was reduced in almost all cases. This varied in degree from a slight reduction to a completely rigid spine, the so-called "poker back". It was noted in the lumbar segment in 96% of the cases, and in the cervical segment in 56%. Spinal tenderness was common in the active stages of the disease. Chest expansion was reduced to less than one and one-half inches in 70% of cases, the majority being under one inch. Not infrequently the thoracic cage was fixed in the expiratory position so that respiration was entirely diaphragmatic.

It should be emphasized that the disease varies greatly in intensity from mild to severe. Spinal deformity may not develop even in cases showing complete rigidity. Activity may cease at any level and progressive involvement of various spinal segments, with a marked constitutional reaction, is seen only in the more severe cases. The percentage that will progress to complete spinal ankylosis is as yet unknown.

A secondary anæmia and slight leucocytosis occurred in about 25% of the cases in this series. An elevated erythrocyte sedimentation rate in excess of 20 mm. (Westergren) was found in 82%. It should be noted, however, that the remaining 18% had a normal rate during the period of observation.

Associated involvement of the peripheral joints, resembling that of rheumatoid arthritis, was observed in 27% of this series (Table IV).

TABLE IV.
PERIPHERAL JOINT INVOLVEMENT IN 100 CASES OF ANKYLOSING SPONDYLITIS

Peripheral joint involvement observed in	27%
Knees	22%
Hips	10%
Ankles	9%
Small joints of feet	8%
Temporomandibular joint	5%
Shoulders	4%
Small joints of hands	4%
Elbows	3%
Sternoclavicular joint	1%

Involvement of the knees was most frequent, occurring in 22 cases; hips, 10; ankles, 9; small joints of the feet, 8; temporomandibular joint, 5; shoulders, 4; small joints of the hands, 4; elbows, 3; sternoclavicular joint, 1. In all these cases there was pain and swelling with or without effusion or radiological evidence of involvement of the joint. An additional 20 patients complained of stiffness and limitation of movements, with or without pain, chiefly in the shoulders and hips, but no structural joint change could be demonstrated. Boland and Present[1] noted typical rheumatoid arthritis of the peripheral joints in 18% of their cases, while an additional 5% had swelling with joint

disability at some other time. Golding[9] reported swelling of peripheral joints in 23% of his cases; Oppenheimer[4] in 18%; Fischer[10] in 29%; and Elliott,[11] in an early review of 50 cases collected from the literature, in 36%.

The first symptoms are not always in the back. In 6 of our cases they were in the chest, chiefly as sternal or thoracic girdle pains. Onset in the peripheral joints, such as the knee, ankle, etc., was noted in 12 cases. Of particular interest was the fact that the disease first became manifest in the peripheral joints in all the cases associated with a preceding acute urethral infection. This peripheral onset in 12% of the cases emphasizes the need for careful spinal examination in all cases of rheumatoid arthritis where latent involvement of the spine easily may be overlooked.

PATHOLOGY

Pathologically the process involves the true diarthrodial joints of the spine which include the sacro-iliac, the small, posterior intervertebral and costovertebral articulations. There is stated to be an early synovitis with small, round cell infiltration and exudation, similar to that seen in rheumatoid arthritis.[12, 13] Later, this is followed by destruction of the articular cartilage and ankylosis. The marked tendency to calcification of the paravertebral ligaments and the ligamenta flava remains unexplained.

When associated involvement of the peripheral joints is present, the clinical and pathological changes are indistinguishable from those of rheumatoid arthritis.[1] This has led some authors[1, 14, 15] to adopt the term "rheumatoid spondylitis" in the belief that the disease is

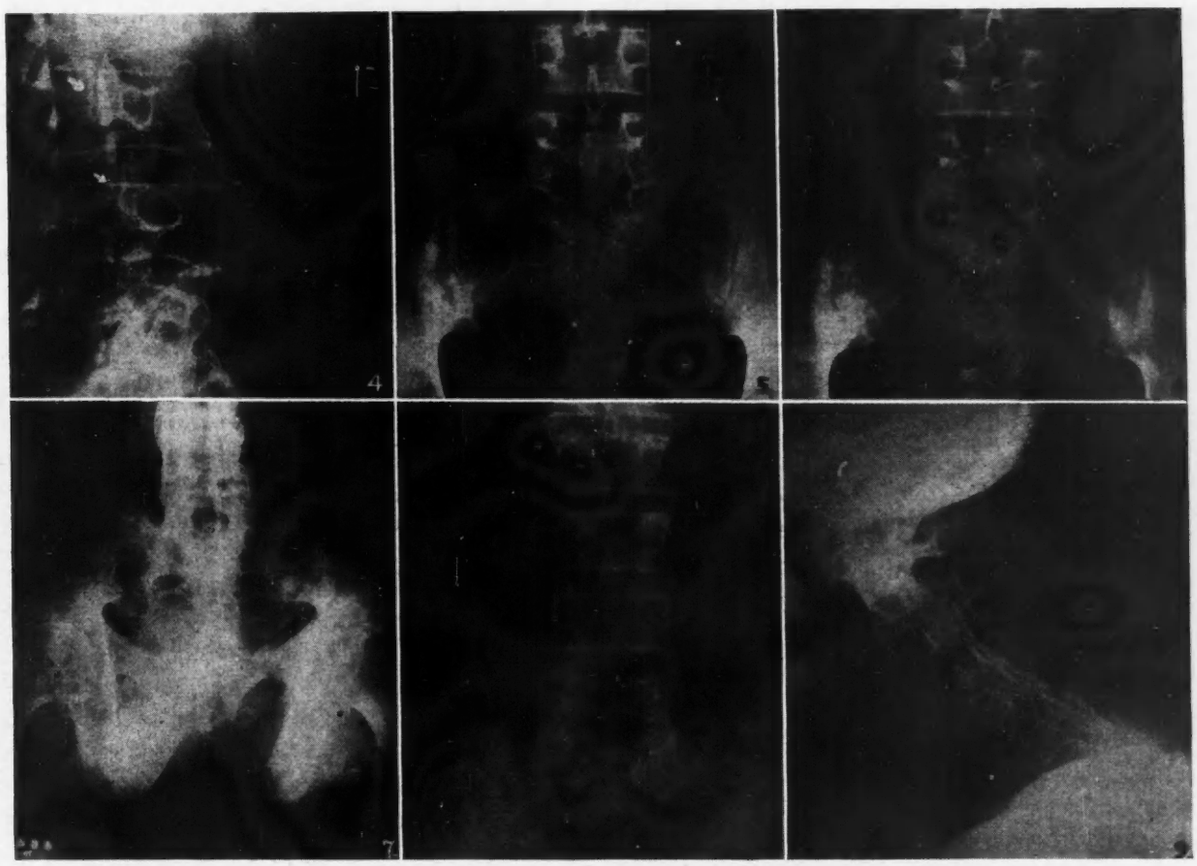

Fig. 4.—Oblique view of the lumbar spine showing irregularity of apophyseal joint margins. There is bony condensation around the joint marked by the upper arrow. **Fig. 5.**—Early case of ankylosing spondylitis. The right sacro-iliac joint shows irregular widening and surrounding bony condensation. The left sacro-iliac joint is relatively normal. **Fig. 6.**—Same case as Fig. 5, one year later. Both sacro-iliac joints are clearly involved. There is no paravertebral ligamentous calcification. **Fig. 7.**—Advanced case of ankylosing spondylitis. (See Fig. 3.) The sacro-iliac joints are completely fused. Ligamentous calcification can be seen in the lower lumbar area. **Fig. 8.**—Advanced case of ankylosing spondylitis (see Fig. 1), with well marked paravertebral ligamentous calcification and obliteration of the sacro-iliac joints (bamboo spine). **Fig. 9.**—Ankylosing spondylitis, showing prevertebral ligamentous calcification of the cervical segment of the spine.

simply a variant of the rheumatoid process. Against this conception is the marked difference in sex incidence, the tendency to ligamentous calcification, the absence of streptococcal agglutinins in a large percentage of cases,[16] the infrequency of subcutaneous nodules of the rheumatoid type,[6] and the lack of response to gold therapy in ankylosing spondylitis.

ROENTGENOLOGICAL FINDINGS

There has been much controversy as to whether roentgenographic evidence of the disease may be detected earlier in the sacro-iliac or in the apophyseal joints. Oppenheimer[4] has made an extensive study of the apophyseal joints and concluded that these show the first evidence of the disease radiologically (see Fig. 4). Involvement of these articulations was demonstrated in all of 50 cases studied, while in 7 cases (14%) the sacro-iliac joints were normal. However, he states: "Since the facets of the articular processes vary in direction, the findings have to be verified in many cases on roentgenograms repeated at slightly different angles. Deviations from the angle required in the individual case may result in appearances falsely suggestive of ankylosis. Owing to the general limitations of roentgenographic interpretation, some findings remain ambiguous even after several examinations."

The more generally accepted view is that the disease commences in the sacro-iliac joints,[1, 5, 9, 17, 18] sometimes being present for several months or years before the spine is involved (see Figs. 5 and 6). Forestier[19] found that the interpretation of changes in the apophyseal joints was unreliable and concluded that most information was obtained on a routine frontal view of the pelvis rather than from special oblique positions. He described three stages in the involvement of the sacro-iliac joints: (1) pseudo-widening of the joint space, first seen in the lower segment of one or both joints with haziness due to marginal decalcification; (2) pycnotic formation in the joint area with the development of a mottled appearance of the cancellous bone on a wide area of the alæ of the sacrum and ilium. This he attributed to decalcification and hypercalcification, the joint space hardly being visible through the mottled bone; (3) terminal ankylosis, usually accompanied by changes in the spinal and iliolumbar ligaments (see Figs. 7, 8 and 9).

TABLE V.
LABORATORY FINDINGS IN 100 CASES OF ANKYLOSING SPONDYLITIS

Radiological evidence of sacro-iliac disease	100%
Calcification of paravertebral ligaments	42%
Increased erythrocyte sedimentation rate	82%

The sacro-iliac joints were involved in all cases in the present series, and there was calcification of paravertebral ligaments in 42 cases (Table V). A variable degree of osteoporosis has been observed, and in a few cases this was a striking feature with marked bulging of the intravertebral discs and central narrowing of the adjacent vertebral bodies.

TABLE VI.
DIAGNOSTIC SUMMARY: 100 CASES OF ANKYLOSING SPONDYLITIS

Males, 94 cases	Females, 6 cases
Young individuals: average age at onset, 24 years	
Pain in the back (early)	82%
Reduced spinal mobility	96%
Elevated erythrocyte sedimentation rate .	82%
Sacro-iliac arthritis	100%

SUMMARY

A review of admissions to an Arthritis Centre (Table VI) has revealed an unsuspected frequency of ankylosing spondylitis and a lack of appreciation of its early diagnostic features.

The ratio of one admission with ankylosing spondylitis to approximately three with rheumatoid arthritis was unexpectedly high.

Possible precipitating factors included: trauma, exposure and urethral infection; but in the majority of cases none was elicited.

The early clinical manifestations and significant radiological changes have been discussed.

Ankylosing spondylitis should be suspected in young adult males complaining of stiffness and back pain with reduced spinal mobility and an increased erythrocyte sedimentation rate. The finding of bilateral sacro-iliac arthritis on x-ray examination confirms the diagnosis.

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RÉSUMÉ

Passant en revue les malades admis dans un centre pour l'étude et le traitement de l'arthrite, on eut la surprise de rencontrer un nombre insoupçonné de malades atteints de spondylite ankylosante; on souligna la négligence d'observer comme il convient les signes précoces qui eussent dû orienter le diagnostic. Sur quatre malades traités pour arthrite rhumatoide, un souffrait de spondylite ankylosante. Les facteurs prédisposants tels que le traumatisme, l'exposition au froid et l'uréthrite ne furent que rarement notés. Les symptômes cliniques du début et l'interprétation des clichés radiographiques ont été longuement discutés. On doit soupçonner la spondylite ankylosante chez les jeunes gens qui se plaignent de raideur et de douleur vertébrales accompagnées de limitation des mouvements de la colone vertébrale et qui présentent une augmentation du taux de sédimentation des hématies. Un radio révélant une arthrite sacro-iliaque bilatérale confirme le diagnostic. JEAN SAUCIER

APICAL LUNG TUMOURS*

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AS early as 1838 Hare[5] described a case of apical lung tumour. The first to draw radiological attention to tumours in the apex of the lung was Pancoast[10] who reported a series of 3 cases in 1924. He was impressed primarily by the nervous symptoms produced, pain in the shoulder and arm and Horner's syndrome. In 1932, he reported 4 more cases and designated them "superior pulmonary sulcus tumours", because of their locality.

Up to the present date about 60 cases have been reported in the literature: by Evans, who discussed Pancoast's original paper, and more recently by Stein,[14] Schaffner,[12] Graef and Steinberg,[4] Jacox and Baker,[7] Henderson,[6] Habein *et al.*,[5] Marcil and Crawford,[8] Owen *et al.*[9] and others. Very recently Ray[11] has made a very complete and concise study of apical tumours, reporting 5 cases.

PATHOLOGY

Pancoast originally suggested that these tumours were pleural endotheliomata. Later, on reviewing the slides of an original case, he changed to spinocellular carcinoma and thought the origin might be in an epithelial embryonic rest. However, he added a rider to this statement; that the idea might again be changed with a better knowledge of the histopathology of the growth. It is now thought that most of them are bronchial carcinoma.

In discussing Pancoast's paper Evans suggested that any type of tumour in the same location would produce the same symptom complex. To support this statement he reported 5 cases which were histologically all different. These cases were: metastatic carcinoma from primary breast cancer; thymic sarcoma; cancer of lung apex; sarcoma of lung apex; sarcoma underneath the scapula.

Of Ray's 5 cases one was metastatic from primary carcinoma of the pancreas and another was a neurogenic fibrosarcoma. His opinion, based on his own cases and a review of the literature, was that the majority of cases were primary pulmonary carcinoma. Owen *et al.*, are of the same opinion. It appears to be generally agreed, however, that it is the situation of the tumour, not its histopathological type, that is of prime importance in producing Pancoast's clinical syndrome.

Pancoast noted a lack of metastases demonstrated radiologically. Steiner and Francis, however, report metastatic deposits in various localities such as; mediastinal and peribronchial lymph nodes, infra- and supra-clavicular nodes, pelvis and right kidney. Fried and Jacox reported metastases in both adrenals.

SYMPTOMATOLOGY

The symptom complex of shoulder and arm pain, and Horner's syndrome (miosis, ptosis, enophthalmos, anhydrosis, hyperæmia and high cutaneous temperature, on the affected side) has been fairly constant to a greater or lesser degree in the cases reported. These manifestations are, of course, due to irritation and involvement of the brachial plexus, upper intercostal nerves and the cervicothoracic sympathetic chain.

* Read at the Seventy-seventh Annual Meeting of the Canadian Medical Association, Section of Radiology, Banff, Alberta, June 14, 1946.

Due to brachial plexus involvement there are motor and sensory disturbances in the shoulder and arm. These are manifested by pain, usually severe, of the arm and shoulder and atrophy of the hand muscles because of ulnar nerve involvement.

The upper dorsal segment of the sympathetic chain supplies the face and eye and destruction of this chain results in Horner's syndrome: paralysis of dilator fibres or iris, miosis; paralysis of superior tarsal muscle, ptosis; paralysis of retro-orbital muscle, enophthalmos; anhidrosis; hyperæmia and high cutaneous temperature.

Due to pressure of the tumour there may be additional signs and symptoms such as hoarseness, dysphagia, œdema of the neck and shoulder due to jugular compression and tracheal displacement to the opposite side. Pulmonary symptoms such as cough etc., are unusual.

As Pancoast suggested, these symptoms simulate those caused by spinal cord or meningeal tumours, neck tumours, cervical rib and vertebral tumours. To this list Habein, Miller and Henthorne add, aneurysm, enlarged lymph nodes, mediastinal tumour, tuberculosis and trauma.

ROENTGEN DIAGNOSIS

Pancoast describes the roentgenologic appearance as being always typical. (1) Apical shadow: This may be so small that it is easily overlooked or mistaken for a small area of fibroid tuberculosis. (2) Destruction or erosion of the upper ribs: this may occur from 3 to 9 months after the onset of symptoms. Steiner and Francis report a typical clinical case with no rib erosion. (3) Destruction or erosion of upper dorsal transverse processes. (4) Occasionally erosion of the sides of the upper dorsal bodies. (5) Displacement of the trachea and related structures to the opposite side.

TREATMENT

Surgical.—Surgical extirpation of the tumour would appear to offer the best chance of cure if the disease has not advanced too far. The results reported have not been very encouraging.

Roentgen treatment.—Like the majority of primary lung cancers, x-ray therapy is not very successful but may produce palliation. One of our cases, although not typical, is alive and well after 18 years, although the tumour is apparently not reduced in size.

Palliation.—Because of the severe pain chordotomy may often be necessary. Opiates in ever increasing doses are necessary in the end stages. In one case, we have been giving 150 c.c. of a 30% alcohol saline solution intravenously with marked relief of pain.

CASE 1

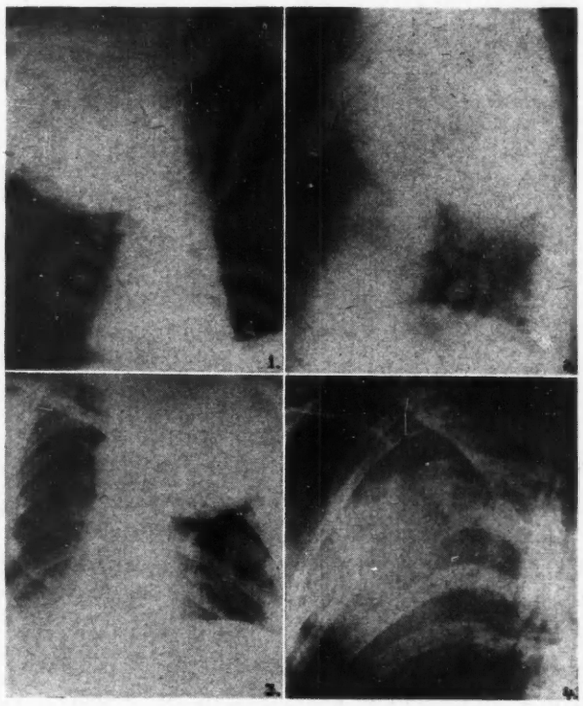
Male, white, aged 32 years. First seen in September, 1921. This patient complained of pain in the left shoulder and arm, present for the last 3 months. The pain had increased in severity until he was unable to sleep.

X-ray film of the chest (Figs. 3 and 4) showed a large dense tumour occupying the upper portion of the left chest. No rib or vertebral erosion, but slight scoliosis. Fluoroscopic and lateral views suggested mediastinal origin.

This patient was seen in the Mayo Clinic in October, 1921. Biopsy from enlarged axillary glands merely showed inflammatory change. He was given x-ray and radium treatment over the chest, axillæ, groins and abdomen, and received further deep x-ray therapy to the chest in 1922, 1923 and 1924 under my direction. Shortly after his first series of treatments, the pain in the shoulder and arm decreased and finally disappeared.

This patient was examined by me in February, 1939 and although chest films showed no change in the size of the tumour, he was perfectly well clinically and was carrying on his trade as a barber.

The opinion from the Mayo Clinic was that this tumour was probably a very fibrous type of lymphoblastoma.



CASE 2

Female, white, aged 49 years. Admitted to Royal Alexandra Hospital June 26, 1937.

History.—Patient unintelligent and speaks little English. Complains of pain in the right chest, shoulder and arm for one year. Occasional cough, no expectoration. Pain has become very severe in the last two weeks. Has lost an indefinite amount of weight.

Examination.—Dullness and marked tenderness over the right upper chest posteriorly. There was wasting

of the hand muscles. The skin of the shoulder and arm was dry. The right pupil was contracted; there was ptosis and evidence of enophthalmos.

X-ray examination (Figs. 1 and 2) showed a dense tumour mass in the upper right chest with erosion of the 4th and 5th ribs posteriorly, and lateral sides of the vertebral bodies. X-ray therapy was given from June 29, 1937, to July 27, 1937. A total dose of 7,200 r (with scattering) through 4 portals was delivered using the following factors, 200 Kv. 5 Ma. 50 cm. T.S. distance ½ Cu. 1 Al.

This patient experienced marked relief of symptoms as early as 3 days after commencing roentgen therapy. Films showed a definite decrease in the size of the tumour and beginning recalcification of the destroyed ribs.

She was re-admitted October 12, 1938. All her symptoms had returned with increased severity. In addition there was œdema of the right supraclavicular and cervical regions, due to compression of venous return. X-ray therapy was again instituted. She received a total dose of 4,220 r (with scattering) and obtained marked relief. The tumour also was reduced in size. This patient lives in a sparsely settled community, a considerable distance from the city and it was very difficult to keep in contact with her. She died in June, 1939.

COMMENT

This is a typical case of superior pulmonary sulcus tumour as originally described by Pancoast. Although there was no biopsy, the tumour was probably a primary carcinoma, moderately radio-sensitive.

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The bright greenish-yellow fluorescence of nails under Wood's light may be utilized to determine qualitatively the ingestion of quinacrine hydrochloride (atabrine). Julius E. Ginsberg, M.D., and Col. Paul L. Shallenberger, M.C., A.U.S., of Chicago state that this characteristic fluorescence appears on the finger- and toenails of those who are on atabrine therapy or atabrine antimalarial prophylaxis or who have been taking the drug within the past six months. Results of this test indicate that complete body excretion of atabrine is delayed longer than formerly reported. Subjects taking only small quantities of atabrine do not fluoresce; those who have not taken atabrine for at least a month show white fluorescence, corresponding to nail growth, in the proximal nail and greenish-yellow fluorescence distally.—*J. Am. M. Ass.*, **131**: 808, 1946.

THE EFFECT OF AMINO-ACIDS ON GASTRIC ACIDITY*

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TO those who believe in the no acid no ulcer theory, it is assumed that any treatment of peptic ulcer to be effective either medically or surgically must cause a marked reduction in gastric acidity. With the introduction of the amino acid treatment by Co Tui,[1] it seemed advisable, therefore, to determine its action in the stomach.

In his first publication, Co Tui stated that gastric analysis had been performed on 18 cases but he reported the results of three only. These showed that a single dose of 50 gm. of amigen reduced the free acid to zero for two hours with a subsequent rise higher than the initial values. Repeated doses of 25 gm. after the initial drop resulted in very much higher acidity.

The purpose of this investigation was to test the effect of varying doses of an amino acid preparation on the gastric acidity in a group of patients whose response to a regular test meal was already known.

METHODS

In all, 16 patients were examined and they may be divided into three groups. Group A—five apparently normal cases, *i.e.*, no gastro-intestinal complaints. Group B—three with digestive symptoms but negative x-ray findings. Group C—eight ulcer cases with positive clinical and x-ray findings, 7 duodenal ulcer and one combined gastric and duodenal ulcer.

The routine procedure was as follows: the patient was given a regular gastric analysis using the Ewald test meal and the acidity computed at 15 minute intervals for three hours. Twenty-four or forty-eight hours later this test was repeated and immediately after the first specimen was withdrawn, *i.e.*, 15 minutes, the dose of amino acid was given by mouth and subsequent withdrawals made at 15 minute intervals. With most patients a second or third dose was given at hourly intervals. The figures for total acidity being invariably high after feeding the amino acid are omitted for the sake of clarity. The pH values were also determined

* From the Gastro-intestinal Clinic, Department of Medicine, Royal Victoria Hospital, Montreal.

throughout but are omitted because they followed closely those of the free acid. The preparation used was quite palatable even in large amounts and we did not encounter any nausea or vomiting during the tests.

RESULTS

Group A.—Normal cases. Figs. 1 to 3 show the graphs of 3 of the 5 cases in this group.

Fig. 1 demonstrates the response to a comparatively small dose of the amino acid repeated hourly in a patient whose regular acidity was rather low; Fig. 2, the response in a patient with high acidity to the same dosage; Fig. 3, the response in a patient with no free HCl until after histamine stimulation, 9 gm. having apparently little effect and 20 gm. producing no free acid for a very brief period.

It may well be noted that the normal cases, those with low original acidity, respond to a small dose of amino acid by a brief reduction of acidity to zero followed by a rebound to a figure higher than the original value (Fig. 1). Those with high original acidity showed higher values throughout the test with but little reduction, the rebound figures being quite high

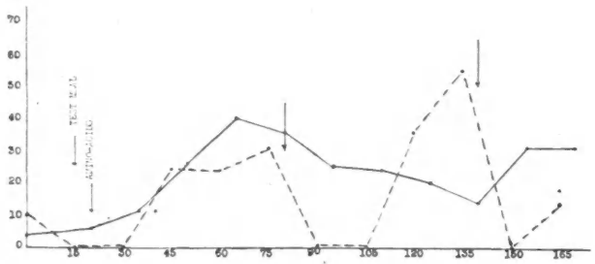

FIG. 1. GROUP A – NORMAL
STRAIGHT LINE – REGULAR TEST MEAL
BROKEN LINE – TEST MEAL PLUS AMINO-ACIDS

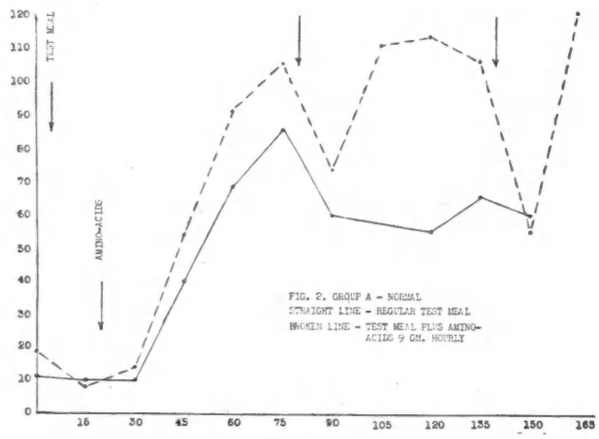

FIG. 2. GROUP A – NORMAL
STRAIGHT LINE – REGULAR TEST MEAL
BROKEN LINE – TEST MEAL PLUS AMINO-ACIDS 9 GM. HOURLY

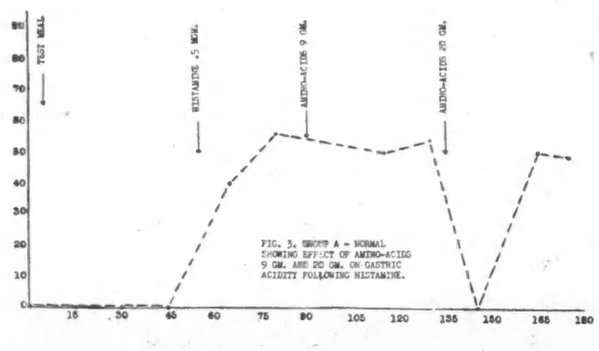

FIG. 3. GROUP A – NORMAL
SHOWING EFFECT OF AMINO-ACIDS
9 GM. AND 20 GM. ON GASTRIC
ACIDITY FOLLOWING HISTAMINE.

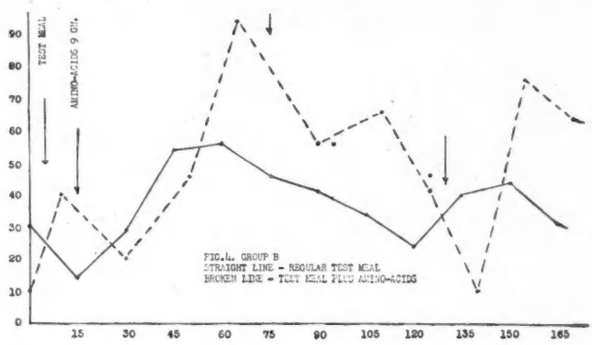

FIG.4. GROUP B
STRAIGHT LINE – REGULAR TEST MEAL
BROKEN LINE – TEST MEAL PLUS AMINO-ACIDS

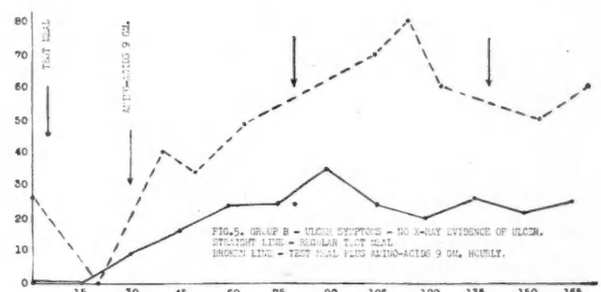

FIG.5. GROUP B – ULCER SYMPTOMS – NO X-RAY EVIDENCE OF ULCER.
STRAIGHT LINE – REGULAR TEST MEAL
BROKEN LINE – TEST MEAL PLUS AMINO-ACIDS 9 GM. HOURLY.

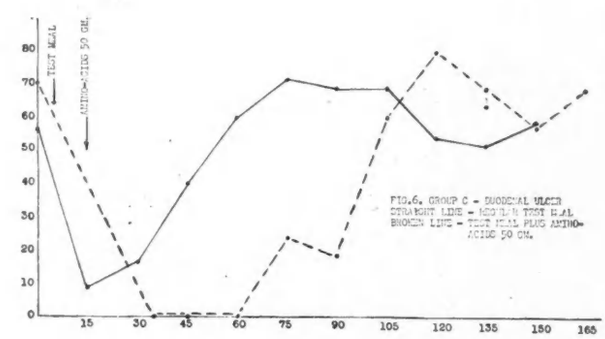

FIG.6. GROUP C – DUODENAL ULCER
STRAIGHT LINE – REGULAR TEST MEAL
BROKEN LINE – TEST MEAL PLUS AMINO-
ACIDS 50 GM.

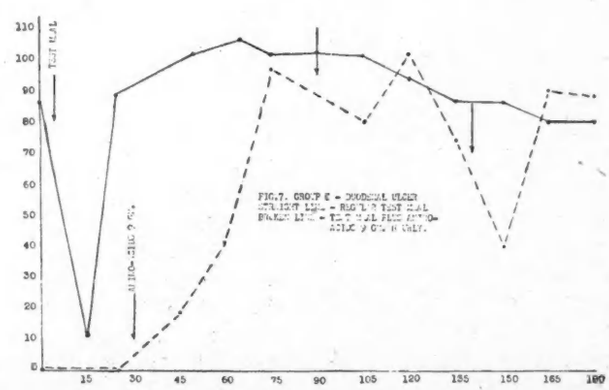

FIG.7. GROUP C – DUODENAL ULCER
STRAIGHT LINE – REGULAR TEST MEAL
BROKEN LINE – TEST MEAL PLUS AMINO-
ACIDS 9 GM. HOURLY.

(Fig. 2). Those with free acid response to histamine showed no reduction after a small dose and a very temporary reduction to zero after a larger dose (Fig. 3).

Group B.—Symptoms but negative findings (functional cases). Figs. 4 and 5 show the graphs of 2 out of 3 cases in this group. Here again the rebound acidity showed higher values than the original.

Group C.—Ulcer cases. Figs. 6 and 7 are the graphs on 2 of 8 ulcer cases, Fig. 6 showing reduction of acidity to zero for 45 minutes after a single dose of 50 gm. (note again the high rebound acidity) and Fig. 7 showing the inadequate response to repeated small doses.

DISCUSSION

In planning this investigation it was felt that by determining the acid curve following a regular test meal and then repeating this test plus amino acid a true picture of the amino acid effect on gastric acidity could be obtained. *In vitro* 5 c.c. of the preparation of amino acid (pH 5) used reduced 100 c.c. of N/10 HCl to pH 5. In the human stomach, however, the results are markedly different. It is evident that in all types of cases, repeated small doses of amino acid cause only a brief reduction of acidity (rarely to zero) but bring about a marked rebound rise of acidity to levels much higher than the original test meal figures. The single large dose of 50 gm. produced a more prolonged reduction to zero but again a secondary rise to high levels.

Thus it would appear as a result of this investigation that repeated small doses of amino acids are of no value in reducing gastric acidity, in fact, they produce a high secondary rise, whereas large doses cause well marked reduction but must be repeated in 1½ hours if the high rebound acidity is to be prevented. These facts are of some importance when one considers that the general public is purchasing amino acid preparations without medical prescriptions and is using doses of six grams or less. Further they confirm Co Tui's[1] finding that repeated large doses are necessary if the treatment is to be effective.

SUMMARY

1. Sixteen patients with normal, functional, or organic disorders of the genito-urinary tract with double gastric analysis were examined first with a regular test meal and then with test meal plus amino acid preparation.

2. Repeated small doses were comparatively ineffectual in reducing gastric acidity and causes a high rebound acidity.

3. Single large doses (50 gm.) produced a prolonged (1½ hours) reduction of acidity to zero but also a high secondary response.

4. It is concluded that amino acids in large doses are of value in reducing gastric acidity but are ineffectual in small doses.

Our thanks are due to Mr. J. H. Hillhouse of the Royal Victoria Hospital staff for his technical assistance.

This publication was made possible through a grant of the Frank Horner Co. Ltd., Montreal, who also supplied the material "Biotol" which was used.

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PAPAVERINE IN THE TREATMENT OF NEUROTIC SYMPTOMS*

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MECHANISMS in the pathogenesis of neurotic symptoms present one of the obvious stumbling blocks to our further knowledge and therapy of the neuroses. There has been a tendency to regard these constantly recurring symptoms as simply "neurotic symptoms" to be used phenomenologically as diagnostic criteria for aiding recognition of the psychoneurotic. Or, in many cases, these are said to be "imaginary" or "psychological", without critical examination of the meaning of these terms, and without carrying the concepts of "imaginary" or "psychological" to any specific conclusion.

The literature on this subject, particularly that in the field of "psychosomatic" medicine, gives much attention recently to the concept of tension or spasm in the production of symptoms of "psychological" origin. In particular, the idea has come to be expressed that in many of the complaints we are dealing with a state of tension of smooth or striated muscle of the various body systems.

Alkan[1] has mentioned the function of contractile elements, consisting chiefly of smooth

* From the Allan Memorial Institute of Psychiatry, Montreal.

musculature, through which he feels that an end-organ reacts to "psychic" influences. He notes that the smooth musculature of the vessels occupies a particularly important place in that it is present throughout the body and affects all the organs by way of its psychically conditioned alteration of function.

Dunbar[2] gives a general consideration of the relation between conflict and muscle tension. The concept of the spasmogenic attitude in which there is a tendency to react by general spasm of smooth or striated muscle or both to any kind of environmental pressure, was introduced by Gallavardin[3] and further discussed by Houston.[4]

Cameron[5] discussing the somatic patterns observed in anxiety states described skeletal muscle patterns and those observable in the cardiovascular and gastro-intestinal systems. Presumably the system patterns may be grouped under the heading of those occurring in smooth muscle.

Kraines[6] in writing of the "physiologic reaction" of the neurotic, says: "the mediating mechanism is the physiologic tension with the resulting increased activity of the vegetative nervous and endocrine systems, its associated tenseness of smooth and striated muscle and the physiologic accompaniment of apprehension and unrest. Such tension is practically always general, although its most annoying manifestation and the chief complaint may be focal."

Given this working concept of spasm or tension as operative in the production of at least part of the somatic symptomatology of the anxiety and tension states, it would appear to be rational to investigate the hypothesis and symptomatic therapy by the use of an antispasmodic drug. The drug selected is the antispasmodic, papaverine.*

PROCEDURE

Six cases have been selected for presentation. In two, the drug was given intravenously for the investigation of a single symptom. In the other four, papaverine was given by mouth over an extended period for treatment of multiple symptoms. At the first visit patients were interviewed, complaints and history recorded, physical and other examinations carried out, and blood pressure and pulse recorded then and at subsequent visits. They were placed upon regular daily dosage of papaverine, and were seen at weekly intervals. The drug was dispensed in ordinary gelatin capsules. At each visit, suggestion was ruled out as far as possible by avoidance of repeated questioning or the asking of leading questions. The patients were encouraged to express themselves spontaneously with regard to any change in their condition. Apart from this, no psychotherapy was done; and no adjuvant, such as another drug, shock treatment, etc., was given.

PHARMACOLOGY

Papaverine is the chief of a group of opium alkaloids which is clinically and pharmacologically distinct from morphine and its related derivatives. In contrast to morphine, papaverine has only insignificant effects on the central nervous system, its main action being an antispasmodic effect upon smooth muscle. It is effective regardless of which division of the autonomic nervous system is overactive.[7] Of its action upon striated muscle, little is known. In a note in his book, Garrod (8) quoting Liedensdorff, says the hydrochloride was found to relax voluntary muscle. Apart from any direct action, we may infer that it would have a relevant effect indirectly in those cases in which it acts upon smaller nutrient vessels that may be in spasm.

The toxicity is low, there is no cumulative action,[9] nor is there any tendency to addiction reported.[10, 11, 12] Its metabolism in the body is not known.[7]

There is little in the literature about side effects or idiosyncrasy in the human. In our patients, we have noticed the following: (*a*) Definite cardio-vascular effect depended upon whether the route was by mouth or by vein. By vein, there followed a definite decrease in the blood pressure, accompanied first by a compensatory rise of the pulse rate, which later fell to below starting level. Oral administration appeared to produce little change in either blood pressure or pulse, save that there was a tendency for widely fluctuating blood pressure as seen in successive visits to become more stable. (*b*) Respiratory effects were noted only upon intravenous injection. There appeared greater depth of respiration with some increase in rate; the patients sometimes complained of breathlessness. The degree of respiratory

* The papaverine used here was supplied through the courtesy of Merck Company.

effect was directly proportional to the rate of injection. (c) With oral administration, two of the patients complained of "sourness of the stomach" or "irritation". This was satisfactorily eliminated by having them take a glass of milk with the drug.

CASE 1

This patient was a 35-year old Jewess suffering from a chronic anxiety state with numerous somatic symptoms; and prominently, a subjective and objective coldness of her hands. This was the particular symptom under investigation.

The patient was first injected with 2 c.c. of distilled water as control. After this she was given 2 c.c. (½ gr.) of papaverine intravenously (see Tables I and II).

TABLE I.

Time	*Temperature*	*Pulse*	*Blood pressure*	*Respiration*	*Remarks*
Before	98.2	74	108/68	22	Hands cool and moist, no headache
5.05		Injection: 2 c.c. Auqa. Dist. intravenously			
5.07	98.2	68	118/82	24	No change
5.12	98.3	68	114/76	22	Hands still cool and moist—fingers quite cool
5.15	98.4	70	110/82	22	Whole hand bilaterally cool and moist
5.25	98.4	70	110/76	20	Palms rather clammy—no change
5.35	98.4	71	108/76	22	No change
5.50	98.3	70	108/74	24	Fingers still cool and moist

TABLE II.

Time	*Temperature*	*Pulse*	*Blood pressure*	*Respiration*	*Remarks*
Before	97.2	68	118/72	20	Hands cold and moist. No headache
4.23		Injection: 2 c.c. (½ gr.) papaverine hydrochloride intravenously			
4.25	98.0	84	112/76	22	Hands still cold and moist. Complains of feeling warmer
4.30					Hands warmer and less moist. Quite red in appearance. Patient feels less warm—but flushed.
4.35	98.2	74	102/68	24	Hands cool but not clammy. No complaints of excess warmth. Hands still appear red about joints.
4.43	98.2	72	104/68	24	Hands warmer and drier. Reddening less marked.
4.53	98.2	71	100/70	22	Hands warmer and less flushed. Palms quite warm
5.08	98.0	68	108/74	22	Face and cheeks quite warm and flushed. Patient notes that she feels "quite relaxed." Hands warm but not sweating.

TABLE III.

Time	*Temperature*	*Pulse*	*Blood pressure*	*Respiration*	Remarks
Before	98.3	72	100/80	22	Numbness about palate and mouth covering upper and lower lips—fairly well circumscribed and consisting of diminished to absent sensation to touch and pain. Also complaints of a generalized "numb feeling" over the whole body. Finger tips cool—rest of hand warm. No sweating.
10.30		Injection: 2 c.c. normal saline intravenously			
10.32	98.2	74	114/84	24	Hands as before. Numbness unchanged.
10.37	98.3	72	114/80	22	No change
10.40	98.2	86	120/78		No change
10.45	98.2	79	114/72		No change
10.48		Injection: 2 c.c. (½ gr.) papaverine hydrochloride intravenously			
10.50	98.2	84	128/72	24	Extent and degree of numbness unchanged. Fingers cool. Rest of hand warm. Says "My body feels warm".
10.55	98.2	71	108/65	22	Numbness as before
11.03		Injection: 3 c.c. (¾ gr.) papaverine intravenously			
11.05	98.3	77	122/68	22	Patient feeling quite warm. Face is flushed. "Body feels warm." Reports that there is a general decrease of "numbness" which has gone over the malar area. Patient complains of difficulty in getting her breath during injection.
11.24	98.3	66	118/72	18	States that numbness has gone from all the body save the upper lip and roof of the mouth. She is still rather pale, pupils three-quarters dilated. Has felt sleepy.
11.40	98.4	65	110/70	16	"It feels as if someone is pouring cold water down my spine." Hands are cold towards fingers. "Roof of my mouth does not feel as numb, but I could bite my top lip off and I wouldn't feel it."
11.48	98.4	65	108/65		"The numbness is coming back in my lower lip again."
12.00	98.4	65	105/65	16	Face has malar flush. Says that she feels more relaxed. Numbness has returned to some extent about the mouth, but malar area and rest of body free.

TABLE IV.

Time	*Temperature*	*Pulse*	*Blood pressure*	*Respiration*	Remarks
Before	98.0	70	110/84	18	Complains of numbness about the mouth (upper and lower lips), cheeks and chin, roof of mouth, and anterior two-thirds of tongue. No other "numbness", hands cool and slightly moist.
11.20		Injection: 3 c.c. (¾ gr.) papaverine intravenously—(in four minutes)			
11.22	98.0	78	114/78		"I feel a bit short of breath." (during injection). Feeling of warmth about the forehead. Numbness unchanged. Hands cool.
11.26	98.1	64	114/70	18	No longer feels warm. Numbness unchanged. Fingers and hands still quite cool.
11.35	98.2	62	108/62	16	Some decrease of hypesthesia about chin. Otherwise unchanged.
11.45		Injection: 2 c.c. (½ gr.) papaverine intravenously—(in one minute)			
11.47	98.1	69	120/74	18	With injection, patient had feeling of considerable warmth all over body. Face is quite flushed over forehead and malar area. Hands are cooler than before. Little change in "numbness". Increased depth of respiration.
11.50	98.0	62	102/68		States that numbness has decreased—is not present about the cheeks or chin. Still present about mouth, roof of mouth and tongue.
11.55	98.0	58	96/66	16	Decrease of hypesthesia on lower lip. Hands are warm and less moist.
12.00	98.1	59	94/66		Further decrease of hypesthesia—gone from lower lip. Hands and finger tips warmer.
12.10	98.1	62	100/78		Says she feels more relaxed. Hands are warm, face is pale with malar flush. Says that numbness is still present in lower lip and roof of mouth, that it is very much less on tongue.
12.20					States that numbness has gone from tongue and roof of mouth. Only present now on upper lip.
12.30	98.2	62	112/68		States that all numbness has gone from areas described before injection. Hands are warm.
12.47					States that numbness has returned to upper lip.

CASE 2

Aged 32. This patient had a chronic anxiety state and a history of many admissions for peptic ulcer. She had had several abdominal operations and was referred because of persistent epigastric distress in absence of demonstrable lesion of the gastro-intestinal tract. In addition to her epigastric symptoms, she presented several other somatic symptoms seen with anxiety; in particular, a persistent "numbness" about the mouth and palate, on the mucous and cutaneous surfaces of the lips. This was the symptom investigated and was described as a "loss of sensitivity" which was a diminished-to-absent perception of touch and pain over areas described. The patient was first injected intravenously with normal saline, then with papaverine (see Tables III and IV).

CASE 3

Aged 37. She complained of headache, loss of appetite, restlessness, palpitations and uneasy sensations in the hands.

She was first given an intravenous injection of papaverine (½ gr.) and appeared to respond favourably. She was then placed on the drug by mouth—1 grain every four hours and on retiring. She showed improvement after two weeks. Subjectively, there was considerably less pounding of the heart, she was sleeping well without sedative, was less restless and her appetite had improved. She continued satisfactorily, and a week before discharge (about 7 weeks after start of treatment) the following note was made: Patient says—"I've never been so relaxed in three years. I would know I had a heart but I can't feel it hardly—before I used to hear it day and night. My head is much better and there's none of that pressure". She had lost the tightening sensation in the throat, had no tremor of the hands or tongue, had lost numb feelings in fingers and body, was eating and sleeping well. There was one of the symptoms which persisted, however—the feeling of paralysis and "deadness inside".

CASE 4

Aged 72. This patient presented the following mixture of complaints: Palpitations and "gas on the stomach"; sleeplessness; poor appetite; restlessness; vague fear of travelling about.

She was placed on papaverine—1 grain every four hours during the day (3 times) and on retiring. After one week, she showed some improvement. She was sleeping better, felt that she was "taking things more quietly", noted that her heart was "not thumping as much as before". Her appetite was not improved. Treatment was maintained and notes made a month later: "I feel better now than I have for years. This last week I haven't been so irritable. My stomach is a lot better and my appetite is quite good. They see a change in me in the house." She was sleeping well. Objectively she was considered more cheerful, less fidgety and restless.

Seven weeks after treatment started, the patient was placed on placebo, consisting of milk sugar put up in capsules identical to those which had contained the papaverine. After a week of placebo, the patient showed return of previous symptoms: "I haven't been well at all this past week. Those pills aren't doing me any good any more." She complained that she could not sleep, that her appetite was poor and that she had a "nervous headache". She said also that she had become irritable, jittery and very restless. Without advice as to the cause of the exacerbation, she was put back on papaverine. In two weeks, she showed improvement: "I've felt much better this week. My appetite has come back and I'm sleeping well again." In one month, she had improved to the extent that she had done before being given placebo. One week later (3½ months after treatment first started) she was again placed upon

placebo, and again there was a definite exacerbation. She was put back on the drug and when last seen was recovering satisfactorily. No attempt was made to wean this patient from the drug, but this was the next step anticipated.

Total duration of treatment was 4½ months during which time the patient received definite benefit. However, it may be noted that she still felt "nervous and jittery" in crowds, walked about outside the house very little, and required someone to bring her to hospital.

CASE 5

Aged 37. Complaints were: (*a*) Frequent crying spells. (*b*) Easy fatigue. (*c*) Loss of appetite. (*d*) Inability to sleep. (*e*) Vague epigastric discomfort. (*f*) Feeling "blue". On examination there were motor signs of anxiety: cold, moist hands with fine tremor of outstretched fingers, tremor about lips when drawn back to expose the teeth, slight tremor of the tongue, a "stiff" gait with shoulders held high in a tense body attitude. Physical examination and barium meal were negative. She was placed on papaverine.

After eight days, patient stated that she is able to sleep better, appetite is improving. She still has the epigastric discomfort of vague nature, has occasional crying spells and frequently feels "blue". She says that she feels less fatigued and appears brighter. Notes two weeks later reported: "The capsules help me I think. My appetite is better." She says that she sleeps through the night and that she is able to work better about the house, cooking and looking after the children. However, she notes that she still feels she is nervous. Extract from notes one month later reads: "Patient states that 'the feeling' in her epigastric region has improved". She is sleeping well and her appetite is good. There have been no crying spells and she says that she feels quite happy. She is also able to do her work at home fairly well and is well enough to look after the children."

The patient continued on treatment satisfactorily for the next two weeks, when she started to complain again of vague epigastric discomfort which was poorly described. This was the only complaint that returned at this time but was persistent. The main trouble seemed to be not so much her other tensional symptoms but the epigastric discomfort which she intimated she felt might be due to cancer. She was given another barium series which was negative. With this reassurance and her general improvement, it was felt that she could continue without papaverine therapy. When last seen seven weeks later, she was still carrying on adequately.

CASE 6

Aged 28. Patient complained of irritability; sleeplessness; palpitations; worrying; listlessness; cold hands and feet. Examination showed manifest anxiety, with tremor of hands, lips and tongue, etc. She responded favourably to a preliminary intravenous injection of papaverine and was placed on oral dosage.

After one week: "Very little improvement. Her hands are warm. Complains of headache which is bifrontal and over vertex." After two weeks: "Patient stated spontaneously that she felt much better. She is sleeping well, has no thumping of the heart. However, she has a headache. She notes that noise does not bother her as much, etc." After three weeks: "She is still sleeping fairly well, and says that she feels very much better. No headache. Decrease in amount of tremor about hands, lips and tongue. She says: I feel more normal now, and I feel I can make decisions. After four weeks: I feel fine and have hardly any complaints at all. I haven't had any headaches and I sleep like a kitten—I seem to be able to relax now. There is still a slight tremor of hands, tongue and mouth. Her hands are warm but moist."

After another week, treatment was withdrawn. During the next week, there was an exacerbation with a return of the majority of her symptoms. She was placed on placebo as in Case 4. During the following 2 weeks, the patient did not improve. She was then put back on papaverine and showed improvement in 2 weeks: "I've been much better this past week. I don't know what it is. I don't feel as jittery and I feel more like my old self." She is sleeping through the night and her appetite is better, etc.

With maintained therapy, she continued to improve and three weeks later it was felt that she was well enough to attempt to take her off the drug. Accordingly the dose was reduced to ½ gr. at the prescribed intervals. There was a slight increase of symptoms during the following week, but this had subsided by the time she had returned for her weekly visit. She was then informed of the plan to discontinue the drug and said she was agreeable, so that treatment was withdrawn. Although there was some return of symptoms, this has not caused the patient any great concern. She has been able to sleep well, has maintained her good appetite, and has felt more relaxed and cheerful. When last seen she had applied for and taken a job in a dentist's office as nurse, had gained weight (twelve pounds and was considering dieting) and was cheerful.

DISCUSSION

A detailed discussion of the results is not possible here, but several of what are felt to be significant points will be touched upon, with consideration of what these appear to suggest with reference to pathogenesis:

The first case shows alleviation of complaint of cold, moist hands. The effects upon the vascular supply of the hands are essentially similar to those observed by Mulinos *et al.*[12] in the treatment of Raynaud's disease by papaverine.

The complaint investigated in the second case was "numbness". This term is vague and in neurotic complaints is on a par with the symptom of dizziness so far as the multitude of meanings that it may have is concerned. In this case, the patient referred to a decreased sensitivity to touch and to pain. In the literature, discussion of disordered sensation in the neurotic refers almost exclusively to the hysteric or to the psychotic. In the case of hysteria, the symptom is postulated to be the result of a dissociation mechanism in which the lower level peripheral sensory mechanisms are unaffected and the failure to register sensation subjectively is due to a blocking occurring at higher or "psychic" levels. For example, working with the cold pressor test, Sullivan[13] has shown that subjective perception of pain and cold are not necessary for the completion of the test and concludes that hysterical anæsthesias do not block stimuli at lowest levels. In discussing the result here, it is felt that the working concept of vascular spasm is the most fruitful. Adams,[14] in discussing the blood supply of nerves, concludes: "Such experimental evidence as is available indicates that its vascularity is of importance to a nerve, and

that cessation of blood supply to any part of a nerve affects the passage of the nerve impulse and will ultimately induce a complete nerve block". It is postulated that increased vascular tension is an accompaniment of anxiety, that this affects the transmission of sensation along the nerves and the drug relieves the hypoesthesia by an antispasmodic effect upon the vessels' spasm, effecting an increased blood flow to the area.

The third case had many complaints. The effects of the drug may be hypothesized as follows: A symptom such as—"from the head down to my shoulders between the shoulder blades, it feels like a jumping toothache"—may be due to tension and fatigue of striated muscle with pain produced either in the muscle itself by accumulation of metabolites, or at the periosteal attachments by mechanical strain. Certain other symptoms are also probably produced by tensional conditions of skeletal muscle with attendant sensations. As noted above, there is little said in the literature about the direct action of the drug upon voluntary muscle. The drug is felt to be effective either in this direct action, or by its relaxing effect upon nutrient vessels that are in spasm as a result of vegetative overactivity, so that increased blood flow would facilitate the removal of metabolites with relief of pain and muscle fatigue, and decrease of irritability. It is recorded that one symptom persisted without change. It is believed with study of psychopathology that this symptom in this patient was symbolic and not tensional. There is not space for full discussion, but it appears that this symptom has reference to a Cæsarean section she has had several years before. At operation, still-born twins were delivered and partial hysterectomy performed. Dynamics of the personality included a neurotic need for affection, with the operation assuming, within her frame of reference, the significance of a traumatic and irreparable loss of fulfilling such need: this loss the symptom symbolized.

In the fourth case, several complaints seem to have been relieved. Of interest here is the effect of withdrawal of the drug without the patient's knowledge: the same capsule filled with substance of identical colour and consistency were supplied. There appeared a definite exacerbation of symptoms which remitted with return to the drug.

The symptoms of the fifth case were vague. It was felt at the time the patient was taken on that this lack of specificity might militate against the usefulness of an antispasmodic. However, there seems to have been some definite improvement. It is postulated that complaints of the type described here are of a generalized tensional nature: that the fatigue is the product of increased muscle tension complicated by poor blood supply to musculature after the fashion suggested in discussion of the third case. The fifth case is typical of a large group of patients who, instead of focal tensional complaints, display a generalized tension spread through the whole musculature. They walk in a stiff, unrelaxed manner and their movements lack the relaxation and co-ordination of the normal. The epigastric complaint appears to have been symbolic; although it is probable that there was an associated hyperactivity of the gastro-intestinal smooth muscle.

The last case demonstrates again the effect of giving placebo as control during the course of therapy.

In conclusion, we have had no patients develop a craving for or addiction to the drug; nor have there been any but mild side-effects or idiosyncrasy.

SUMMARY

1. Working hypotheses concerning the relationship of muscular spasm to neurotic complaints are described.

2. Papaverine hydrochloride (an antispasmodic) was selected as a means of investigating this relationship.

3. A large range of complaints, including numbness, coldness and sweating of the extremities, generalized tension, etc. was relieved. In addition, patients' concern over these complaints, as well as the general feelings of anxiety, were relieved.

4. Discussion is made of grounds for suggesting that certain of the "imaginary" somatic complaints of psychoneurotic patients are due to spasm of smooth musculature—in particular, that the spasm of smooth musculature of vascular walls throughout the body is of paramount importance in this regard.

5. A distinction is drawn between *tensional* and *symbolic* complaints.

6. While alleviation was obtained, the chief value of papaverine appears to be as a research tool, and results encourage further work in this

direction for the elucidation of both pathogenesis and symptomatic therapy by the use of this and other antispasmodic agents.

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RÉSUMÉ

Les relations possibles entre les spasmes musculaires et les symptômes névropathiques servirent ici d'hypothèse de travail. Le produit employé pour l'expérimentation fut le chlorhydrate de papavérine, un antispasmodique bien connu. Un grand nombre de malaises subjectifs ont été ainsi améliorés. On croit que certaines manifestations névropathiques peuvent être sous la dépendance de spasmes de la musculature lisse. On distingue des malaises *tensionnels* et des malaises *symboliques*. Les résultats obtenus doivent encourager des recherches ultérieures afin d'élucider les mécanismes pathogéniques profonds des divers états de *tension* observés en psychiâtrie.

JEAN SAUCIER

NUTRITIONAL DISTURBANCES AND THE ORAL USE OF AMINO ACIDS IN INFANTS*

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IT is a well known fact that severe nutritional disturbances appear chiefly in artificially fed infants, as opposed to those who are breast-fed. Moreover, these disturbances are almost a logical consequence of artificial feeding and, to a certain extent, in each case can be foreseen, if not actually expected. Many aspects of the development of pædiatrics are due to the widespread practice of artificial feeding.

In the beginning, medical science concerned itself mainly with the digestive effects of such feeding. However, it was not long before the discovery was made that a broader view must be taken, so that the more remote influences would be included. The initial step was to correlate digestion with nutrition and then nutrition to the integrity of the human being as a whole. It is almost unnecessary to name these influences, although the more important ones ought to be kept in mind. For instance, the relationship between standard physical measurements and physical strain or aptitude; resistance to infection; the conservation or acquisition of immunity; the integrity of organs and their functions; mental attitudes; intellectual effort, etc.

At first also the medical profession was concerned with the bacteriological aspect of the problem. Even though this was a necessary stage in the growth of our knowledge, for it controlled many causes of death and emphasized the importance of vitamins, yet we are bound to admit that it helped to conceal for too long, another important aspect; that of the biochemical ill-consequences. Medical instinct in this is almost as old as the world itself. Proof of this can be readily advanced when one remembers the painstaking efforts applied for the purposes of human milk conservation and its emergency supply. And also, by the striking results obtained when employed in serious conditions. Were they agreed upon for the sake of bacteriological safety alone, or for that safety increased to its maximum by biochemical adaptation?

Thus convinced and prepared, pædiatricians were in great need of pre-digested proteins, such as amino-acids. Even though they were still obtained from artificial sources and were derived from milk casein, yet they seemed to approach the formula nearest to human milk, especially in the case of sick infants whose normal processes were impaired largely through lack of assimilation and by great loss of necessary nutrients.

Since the month of March, 1945, their oral use, in the Pædiatric Department of the Saint Sacrament Hospital has been more or less routine procedure at any stage of what is usually called, toxicosis or severe malnutrition. These names are preferred to about a dozen others, to avoid confusion. They have been selected because they serve to describe best an advanced stage, whether of simple acute

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diarrhœa, severe acute dyspepsia or the choleriform state.

When this procedure was adopted, gain in weight seemed quicker and steadier, clinical signs of intoxication and dehydration, such as the grayish color of the skin and the loss of skin elasticity, diminished noticeably within the first 24 hours and, in many instances, vanished altogether. Stay in the hospital gradually shortened, insofar as toxicosis itself was concerned. Certainly these results were not only clinical impressions. We compared them with those obtained during the preceding two years.

Prior to the amino acid period, in any case of toxicosis our technique was as follows: upon admission, the infant was fed every four hours —except in cases of very severe vomiting— with a certain amount of pectin-agar and dextri-maltose. During that period, and the days following, the infant received, intravenously, liberal amounts of mixed dextrin and saline isotonic solution. At 12 or 15 hour intervals, 10 to 30 c.c., of 20% hypertonic glucose solution, was injected simultaneously with, in emergency cases, small transfusions of whole blood or plasma. When the intravenous route was, for some reason or other, unavailable, the subcutaneous one was resorted to, restricted to normal saline solutions, with meagre results. When the child showed improvement, buttermilk or skimmed acidified milk was progressively added to the formula. With slight individual variations, such procedure was followed until March, 1945. Since then, however, although the same procedure was followed, the kind of milk used was replaced with amino acids and the following results were obtained.

TABLE I.
NUMBER OF DEATHS

Dates	*Total No. of cases*	*Total No. of deaths*	*Percentage*
March, 1943 to March, 1945	31	23	74.2
March, 1945 to March, 1947	40	18	45.0

The total number of deaths, for the first two years, should read 14 (45%). This because a few cases spent only one day in the hospital, or died in a toxic condition because of disease, such as Von Gierke's disease, atalectasia, pleurisy, etc. For the same reason, the last two years should read 10 (25%).

TABLE II.
NUMBER OF CURES

Dates	*Total No. of cases*	*Cures with amino acids*	*Cures without amino acids*	*Percentage*
March, 1943 to March, 1945	31	..	8	25.8
March, 1945 to March, 1947	40	17	5	55.0

The percentage of cases cured without the use of amino acids during the last two years is insignificant inasmuch as it bears a relationship of only 12.5% to the total number of cases.

These figures definitely speak for themselves. They indicate the practical value of the oral use of amino acids. The child was given a daily dose of from 10 to 12 grams (3 to 6 teaspoonfuls per bottle). It did not take long to discover that this controlled vomiting and diarrhœa better than hitherto and was well tolerated. It seemed to help the organism fight infection to an unexpected degree. For example, many infants were admitted who had no fever, and after two days of treatment fever developed and an infectious state superseded the toxic condition which was greatly benefited by the use of penicillin or any of the other anti-infectious agents. The child seemed to respond more readily to the use of these agents after amino acids were used. The renal function of the child also benefited. The urine increased in quantity and casts and cells gradually disappeared. The urea content of the blood, in certain cases, rapidly decreased to normal figures. No other chemical findings are mentioned here because they have the same meaning as the urea content of the blood. They indicate an actual finding of scientific value but not of great clinical importance. Such findings are important in hospital routine (or were important), but as the information they give has been restricted to a more or less absolute meaning, their clinical value has decreased, particularly in infancy.

CONCLUSIONS

1. The oral route for the administration of amino acids seemed, in our experience, to comply with the requirements of sick infants.

2. This route can easily be used in home practice, when hospital routines are unavailable, and when the practitioner needs a precious time-saving technique.

3. The biochemical restoration of the organism is obtained through natural channels,

which does not contradict our previous knowledge concerning the treatments of toxic nutritional disturbances in infancy. Rather a better result is permitted when both are simultaneously applied.

4. Clinical harmlessness, and nutritional value, give to amino acids an unsurpassed practical value.

5. Their action does not seem to be limited to the digestive system alone. A favourable action on the course of parenteral infection results as well.

6. Clinical symptoms of acidosis, ketosis or uræmia are a sufficient indication by themselves.

7. Any blood information has been, to a certain extent, intentionally neglected, because, although precise, it cannot include tissue biochemical condition.

SUMMARY

Since March, 1945, the oral administration of amino acids, in toxic conditions of infancy, combined with routine treatment has been of great practical value. Cases have been studied for two years prior to their use and during the two years since their use. Results obtained in our cases seem to favour them as a routine procedure.

The amino acids referred to are Mead Johnson's product, "nutramigen".

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Rectal bleeding is a serious warning. Assumption that the cause is hæmorrhoids, without examination, may permit an undiscovered carcinoma to progress to an inoperable stage. Yet 60% of malignant lesions in the terminal portion of the colon are within easy reach of the examining finger. Symptoms of malignant tumour are often ascribed to colitis, dysentery, or chronic diarrhœa.—Louis J. Hirschman.

INTERPRETATION AND SIGNIFICANCE OF FALSE POSITIVE SEROLOGIC REACTIONS FOR SYPHILIS*

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BIOLOGICAL false positive tests for syphilis are positive Wassermann or flocculation reactions which are not caused by syphilis, and are not due to clerical or technical errors, or faulty materials. They are caused by diseases other than syphilis, not necessarily infectious diseases, and are found even in individuals with no evidence of any pathological state. They may be caused by antibodies similar to the syphilitic ones or by some as yet unknown changes in the composition of the human serum.

A short time after Wassermann, Bruck, and Neisser had introduced the complement fixation test for syphilis, it was found that a number of other diseases also gave a positive reaction in a more or less high percentage of cases. However, as long as Wassermann reactions had been done in suspected cases only, the danger of biological false positive findings was unimportant. When, however, tests for syphilis became routine procedures in many hospitals and doctors' offices, and when large groups of individuals such as young men being inducted into the armed forces and couples about to be married were examined, it became obvious that many conditions other than syphilis gave false positive complement fixation or flocculation tests. Therefore, it is only in recent years that interest in these false positive tests has been aroused.

The list of diseases and conditions which may produce false positive reactions is still growing. The following table, indicating many of the conditions in which false positive reactions are known to occur, includes only the more common conditions.

The percentage of false positive findings varies widely for the same disease, according to different authors. This may be explained by the use of different tests, by a difference in the numbers of tests made in a single case, and the period which has elapsed between the onset

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TABLE I.

DISEASES AND CONDITIONS WITH FALSE POSITIVE SYPHILIS REACTIONS

(1) Frequent	
Yaws	Weil's disease
Malaria	Infectious mononucleosis
Leprosy	Typhus
Relapsing fever	Lymphogranuloma venereum
Rat bite fever	Disseminated lupus erythematosus
(2) Occasional	
Scarlet fever	Atypical pneumonia
Measles	Infectious hepatitis
Chickenpox	Vaccination
Chancroid	Immunization with tetanus toxoid
Herpes genitalis	Hæmolytic anæmia with autoagglutination of red cells
Tuberculosis	
Vincent's angina	Malignant tumours
Pregnancy	Menstruation

of the disease and the time the test was performed. Routine tests taken on admission to the hospital may give completely negative results because the test is performed too early. Another series of cases of the same disease may show a high incidence of false positive findings, when the tests are made later in the course of the disease. However, it is beyond the scope of this paper to discuss the details of technique.

Not one of the various types of serological tests which are employed today is free from the objection of giving occasional false positive reactions. Some do it more frequently than others. Certain tests give less false positive results with one disease and more with others. There is no way to decide with certainty by a single serodiagnostic procedure whether a positive reaction is a biological false positive or is a true one, caused by syphilis.

ASSESSMENT OF REACTIONS

What are the ways to differentiate between a false positive and a true positive, between a specific and a non-specific reaction?

The suspicion of the physician should be aroused when an unexpected positive report is received. As always when a positive test is reported for the first time, the test should be repeated at once to exclude technical errors. If the positive finding is confirmed, a careful history should be taken with special reference to symptoms of syphilis, to possibilities of exposure as well as to known causes of non-specific reactions. Any febrile disease or active immunization within six months preceding the test may account for a false positive reaction. A careful physical examination should follow, searching not only for clinical signs of syphilis, but also for non-syphilitic diseases known to give false positive reactions.

However, history and clinical examination will bring conclusive proof for or against syphilis only in a very limited number of cases. Every physician knows how little he can trust a negative history in a syphilitic patient. The value of clinical investigation in such doubtful cases is not high either. Stokes and his co-workers found that even a board of experienced specialists could not make a final decision upon clinical investigation, when called upon in a large series of persons who had been found to have a positive blood test on occasion of blood donation for the Red Cross.

In this connection it is rather interesting to note that lymphogranuloma venereum, chancroid and herpes genitalis have a high incidence of false positive reactions. For obvious reasons each of these diseases is likely to deceive the physician into the assumption that the positive report, even if not 4+, will mean early syphilis.

Blood tests of other members of the family or of sexual contacts may sometimes be helpful. Congenital lues can almost certainly be ruled out if the parents and their other offspring are negative. A negative test on the blood of the sexual partner is some evidence against syphilis. However, here too the findings are not a 100% proof, since the disease may have been cured or burned-out and so give a negative reaction.

DETECTION OF NON-SPECIFIC REACTIONS

The two most important steps which must be taken in order to detect cases of non-specific positive syphilis reactions are as follows: (1) the withholding of antiluetic treatment until a final decision is reached. Treatment may be delayed as long as six months and even longer. Most of the false positive reactions become negative within a few weeks. If treatment is started at once, a true diagnosis may be obscured or made impossible, since it cannot be decided if the reversion of the reaction after a few injections of antiluetic substances is produced by the treatment or is due to the fact that the patient never had lues. There is only one exception to this rule and that is pregnancy. Here the risk for the expected infant becomes too great after the fourth month of pregnancy to justify the withholding of therapy in case the condition turns out to be lues.

(2) Repetition of the serological tests every 2 to 4 weeks. As already mentioned, most of the non-specific reactions become negative in a short time. The laboratories should be asked to use all the various methods at their disposal; and the same blood sample should be sent to two or more laboratories. A complement fixation test should be included in the list of tests. Discrepancies in the tests and discrepancies between different laboratories are unusual in untreated syphilis. If the reaction is strong enough, titration should be done regularly. False positive tests become lower in titre, whereas untreated syphilis would not show such a decrease.

Testing of the spinal fluid in cases without syphilitic symptoms can be postponed until all other tests have been made, *i.e.*, until the end of the waiting period. A negative finding in spinal fluid is no proof against syphilis. A positive finding is usually regarded as proof for syphilis. However, from a theoretical point of view the possibility of false positive reactions in spinal fluid cannot be denied. For obvious reasons systematic studies of spinal fluids in sufficient number have not yet been made. Indeed few observations of certainly false positive reactions to Wassermann tests are published. Scott, Reynold and Mohr have reported 7 cases of meningitis of varying causation which gave several positive spinal tests before reverting to negative. Two cases of encephalomalacia and one case of meningococcal meningitis have been published by McLean and Munger, and by Davis respectively.

A few case reports may illustrate the more or less academic arguments. All the cases have this in common: the serological findings were accidental. The tests performed comprised complement fixation with alcoholic and cholesterol extracts, Kline diagnostic and Kline exclusion and Kahn test. The strength of each test was 3 or 4 plus. There was no history or clinical evidence of syphilis.

CASE 1

This was a woman seen because of menopausal complaints. Blood tests of the husband and two children were negative. The reactions of the patient remained positive in full strength for six months; after that period they became negative and remained so, proving that this was a case of biological false positive reaction.

CASE 2

This was a young woman in the second month of her first pregnancy. The husband's blood was negative. Because of the pregnancy, treatment was started after the third month. This case is still under observation.

CASE 3

This was a woman under treatment with high doses of penicillin because of pelvic peritonitis. A few weeks after treatment was finished the reactions became negative, leaving the question open, whether patient did or did not have syphilis. To know it is important, since the treatment cannot be regarded as sufficient in case of lues.

It is by no means the intention of this presentation to shake the physician's confidence in the serological reactions for syphilis. However, every physician should be aware of the fact that serological tests for syphilis are subject to errors of a biological nature and that a serum positive to all different serological tests may not mean syphilis.

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INFLUENZAL MENINGITIS*

(A report of three non-fatal cases)

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IT is generally recognized that meningitis in infants due to *H. influenzæ* is associated with a high mortality in untreated cases. Treatment with sulfonamide compounds combined with rabbit serum containing antibody against organisms of Type B, which are responsible for the majority of cases, is reported by Alexander[1] to have resulted in a significant lowering of the death rate. More recently Herrell and Nichols[2] have described 4 cases of influenzal meningitis treated successfully with streptomycin, and Weinstein[3] has reported recovery in 7 of 9 cases treated with this antibiotic.

The purpose of this communication is to record 3 non-fatal cases of influenzal meningitis, two of which were treated with penicillin and sulfonamides. The third case, after similar

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initial treatment, received a course of streptomycin therapy.

CASE 1

M.H., a 5 months' old girl, was admitted to hospital on August 16, 1946, under the care of Drs. Urban Gareau and F. C. McCulloch. She had become ill 24 hours previously with the sudden onset of feverishness, fretfulness and listlessness.

Physical examination revealed marked irritability, neck stiffness and a bulging anterior fontanelle. The temperature was 99° F. The urine showed a trace of albumin. The blood leukocyte count was 9,200 with 62% neutrophils, 29% lymphocytes and 1% monocytes. Lumbar puncture yielded a cloudy fluid containing 430 cells per c.mm., 96% of which were neutrophils. Bacteria were not seen on direct examination of a centrifuged specimen but culture yielded *H. influenzæ*.

Immediately on admission, 10,000 units of penicillin were given intrathecally and 20,000 units intramuscularly. Over the 4 following days, further intrathecal injections of penicillin totalled 40,000 units. Intramuscular injections of penicillin were continued every 3 hours for 38 days, totalling 4,200,000 units. Soludiazine 2.5 gm. was given intravenously on the day after admission. On the 4th day in hospital, intramuscular soludiazine 0.4 gm. daily was commenced and continued for 8 days. Sulphadiazine was given later, on the 23rd hospital day for a period of 6 days, because of a rise in temperature and cerebrospinal fluid cell count. Over this period, 0.4 gm. daily was the dosage.

During the first three days in hospital the patient seemed severely ill but thereafter clinical improvement was fairly rapid. The cerebrospinal fluid yielded *H. influenzæ* on culture on the second and third day after admission, and thereafter was repeatedly negative. The cerebrospinal fluid cell count was 7,200 per c.mm. on the second day, of which 98% were neutrophils. The count fell to 3,800 cells on the third day and 508 cells on the fourth. The temperature ranged between 100 and 103° F. for the first week in hospital and then became normal until the 23rd day after admission when there was a rise to 101° F. This coincided with a rise in the cerebrospinal fluid cell count from 77 cells a few days previously to 710 cells. However, there was no growth on culture of the cerebrospinal fluid, and three days later the temperature was again normal and the cerebrospinal fluid contained 100 cells per c.mm.

On October 4, 1946, the infant was discharged from hospital. The cerebrospinal fluid contained 20 cells per c.mm. The blood leukocyte count was 5,050 of which 56% were neutrophils, 38% lymphocytes, 6% monocytes and 1% eosinophils. Cultures of the cerebrospinal fluid had been repeatedly negative since the third day in hospital.

CASE 2

J.S., a 4-year-old girl was admitted to hospital on October 9, 1946, under the care of Drs. W. C. Rennick and J. Lloyd Brown. On the evening of the previous day the child had begun to complain of a severe headache and when first seen was semi-conscious.

Physical examination revealed the child to be comatose, with severe neck rigidity and generalized muscular twitchings. The rectal temperature was 105° F., the pulse 150 per minute and the respirations 48 per minute. The blood leukocyte count was 8,000 with 75% neutrophils, 22% lymphocytes and 3% monocytes. The urine showed a trace of albumin. The cerebrospinal fluid was cloudy and under increased pressure. It contained 6,700 cells per c.mm. of which 94% were neutrophils. Bacteria were not seen on direct examination of a centrifuged specimen of the cerebrospinal fluid, but *H. influenzæ* was isolated on culture.

On admission, 20,000 units of penicillin were injected intrathecally and 25,000 units intramuscularly. Injections of penicillin into the spinal theca were repeated on the three following days, so that the total amount received by this route was 80,000 units. Intramuscular injections of penicillin were continued for five days totalling 1,800,000 units. A daily dose of 1.5 gm. of sulfadiazine was given for the first 2 days in hospital. Soludiazine was administered intravenously and intramuscularly for the first four days, the dosage being 4 gm. a day. On the fifth day in hospital soludiazine dosage was stopped and sulfadiazine by mouth recommenced and continued for 5 days at 3 gm. a day.

The child was obviously most severely ill on admission. On the next day, although the blood leukocyte count was 21,500, the temperature dropped to 100° F. and fluctuated between 100 and 101° F. until the sixth hospital day. After this it did not rise above 99° F. The convulsive twitchings ceased on the day after admission and by the following day clinical improvement was evident. The cerebrospinal fluid cell count reached a peak of 16,100 on the morning of the day after admission, but had dropped to 3,888 cells by the end of the third day, and to 110 cells by the fourth day.

H. influenzæ was obtained on culture of specimens of the cerebrospinal fluid obtained on the first and second days in hospital. Thereafter the fluid was repeatedly negative on culture. By the end of the first week in hospital the blood leukocyte count was 9,200. The child was discharged from hospital apparently perfectly well on October 24, 1946, 16 days after admission.

CASE 3

D.C., a 15-month-old boy was admitted to hospital on September 30, 1946, under the care of Dr. A. S. Sinclair. The child was reported to have been ill for less than 24 hours.

Physical examination showed the patient to be pale and irritable, with a rigid neck and slight opisthotonos. The temperature was 99° F. The urine showed a trace of albumin. The blood leukocyte count was 16,850 of which 78% were neutrophils and 22% lymphocytes. On lumbar puncture the fluid was turbid and contained 6,010 cells of which 85% were neutrophils. Organisms were not found on direct examination of the cerebrospinal fluid but *H. influenzæ* was obtained on culture.

Administration of penicillin intramuscularly was commenced and continued for 5 days, injections being given every 3 hours, over which period 760,000 units were given. In addition, on the third and fourth hospital days 40,000 units of penicillin were given intrathecally. From the second to the fifth day in hospital, sulfapyridine was also given, the dosage being 1.5 gm. on the first day and thereafter 1 gm. a day. On the sixth day after admission, penicillin and sulfonamide therapy was discontinued and a course of streptomycin commenced. An intrathecal injection of 20,000 units was administered and repeated 2 days later. Intramuscular injections were given every three hours for 2 weeks. In all, 3,100,000 units of streptomycin were received.

The child was obviously severely ill on admission and remained so for three days. The temperature ranged between 100 and 102° F. The cerebrospinal fluid cell count was 10,833, with 82% neutrophils, on the third day in hospital. However, on the fourth and fifth days some clinical improvement was noted. The temperature fell to below 100° F. and the cerebrospinal fluid on the fifth hospital day contained only 112 cells. It was on the following day that streptomycin therapy was commenced. This coincided with a rise in temperature to 101° F. A lumbar puncture done 5 days later showed a cell count of 765, 90% neutrophils. However, the child's condition slowly improved. After the first two weeks in hospital the temperature remained normal. The cerebrospinal fluid, on the nineteenth day in hospital showed 166 cells. At this time the blood leukocyte content was 7,500 of which 29% were neutrophils, 68% lymphocytes, 2% eosinophils and 1% basophils. The sedimentation rate was not elevated.

In this case *H. influenzæ* was grown repeatedly on culture of the cerebrospinal fluid. Cultures were positive on the first, fourth, sixth, eleventh, twelfth, and

thirteenth days in hospital, but thereafter were negative. The child was discharged, apparently well, on October 22, 23 days after admission.

COMMENT

Cultures of *H. influenzæ* from cases 2 and 3 were submitted to Dr. F. O. Wishart, Department of Hygiene and Preventive Medicine, University of Toronto. He reported that agglutination reactions indicated both strains to be Type B. The organisms in case 1 were not typed. Sensitivity tests were not done on the strains from cases 1 and 2. The strain isolated from case 3 was sensitive to 200 units of streptomycin per c.c. (4 times the concentration which inhibits strains considered sensitive to streptomycin) only slightly sensitive to 0.1% solution of sulfadiazine and insensitive to 10 units of penicillin per c.c. (10 times the concentration which inhibits organisms sensitive to penicillin).

In the first two cases, treatment with penicillin and sulfonamides was followed by fairly prompt clinical improvement and eventual apparently complete recovery. As both patients were extremely ill on admission to hospital, and as influenzal meningitis is highly lethal in untreated cases in children, it seems possible that recovery in these cases was related to therapy. In the third case, although eventual recovery is attributable to streptomycin, some clinical improvement and a drop in the cerebrospinal fluid cell count occurred during preliminary treatment with penicillin and sulfonamides. However, this treatment did not sterilize the cerebrospinal fluid.

It has been believed for some time that sulfonamide compounds potentiate the effect of penicillin against susceptible organisms.[4] That these therapeutic agents may exhibit synergy against bacteria resistant to the effect of either separately is suggested by the recent work of Bigger.[5] Since *H. influenzæ* is relatively resistant to penicillin and to the sulfonamides *in vitro*, recovery in the first two cases here reported, if attributable to therapy, is suggestive of such a synergic effect.

Our thanks are due to Dr. F. O. Wishart and others of the staff of the Department of Hygiene and Preventive Medicine, University of Toronto, for their assistance.

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BACKACHE AND FIBROSITIS: A MEDICAL POINT OF VIEW

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BACKACHE is so common and the causes are so numerous that the medical journals yearly produce their unfailing crop of articles, each of which reveals its author's favourite theory of cause and treatment. On this side of the Atlantic the more recent articles have been mostly by orthopædic and neurological surgeons who are interested mainly in conditions pertaining to their special fields of study, while from England have come some articles from physicians and physiotherapists on non-surgical causes of backache. For lack of space, it would be impossible to mention even by name all the possible causes of backache but it must not be forgotten that secondary tumours may invade bones, that tuberculosis, even if rare now, may still occur and that intervertebral discs may protrude from their normal resting places. From the point of view of the physician the most common cause of backache is fibrositis with its frequently accompanying muscle spasm. In the practice of the internist at least a quarter of one's patients will show some evidence of these conditions if search is made for them.

Fibrositis is the name given to a condition that produces in certain susceptible individuals small nodules in the connective tissue; these are sometimes painful or may give rise to referred pain or may be only tender to the touch. It is probably the most common cause of pain in the large group of individuals who suffer from so-called rheumatic conditions. This is well shown in a recent article by Savage[1] on Rheumatic Disease in the Forces. Out of 270 men seen at two military hospitals in England with rheumatic pain the cause was fibrositis in 52%.

Some authors speak of fibrositic nodules and others, mostly English, of rheumatic nodules. Mennell[2] writes of sensitive deposits and this rather indefinite term has much to commend it, as a description of the pathological process will show. It will probably be wise to continue to use the term fibrositis and fibrositic nodule, remembering that the implication that an inflammatory lesion is present is not strictly correct.

In the English literature fibrositis is usually grouped with rheumatic conditions, in which are also included various types of arthritis. In this country, however, we would prefer to follow a different classification and keep fibrositis separate from these other conditions, as it has many points of difference. Fibrositis begins most commonly in early adult life, the usual age of onset being younger than that of osteo-arthritis. There is no evidence of infection, as shown by the absence of polymorphonuclear leucocytes in the lesion and by the normal temperature, normal leucocyte count and normal blood sedimentation rate. This is in direct contrast with the conditions in acute rheumatic fever and rheumatoid arthritis. In the literature there is not much mention of sex incidence or age of onset. In my own practice I have found females more frequently affected in the ratio of three to two and although I do not see many children, I have found signs and symptoms at as early as eight years of age.

The predisposing causes have been described by R. G. Gordon[3] as (1) sudden strain, (2) wear and tear, (3) chill and (4) worry, or some combination of these. To these may be added two more causes which seem to be the main ones in a great proportion of people seen here, namely poor posture and lack of use of certain groups of muscles. Under these conditions and in certain people only, the fibrous tissue in the neck, shoulder girdle or upper part of the lateral spinal muscles may react by the development of painful nodules which, after a little practice, may be readily found by palpation. The nodules vary in size from those that are just palpable to others the size of a small pea.

The essential pathology of fibrositis is now well recognized, as the nodules at various stages have been examined microscopically following excision. Stockman[4] has described the changes in the white fibrous tissue of the muscles, nerves and fascia as being due to œdema with sero-fibrinous exudation; in this the fibroblasts proliferate rapidly, numerous minute new blood vessels appear and the whole forms a soft ill-defined congested little swelling. In the exudate polymorphonuclear leucocytes are entirely absent with the consequent absence of pus formation. At this stage the small nodules may resolve rapidly under treatment or spontaneously but more frequently they increase in size from continued proliferation of their connective tissue cells, thereby becoming more fibrous and tougher.

Sir Thomas Lewis[5] has described the pain of acute fibrositis as of the same nature as visceral pain. He considers that muscular pain which is of the deep structure variety is referred to a different area of the sensorium from skin pain. Such pains are often associated, as are those from the viscera, with a slowing of the pulse, a fall of blood pressure and nausea; the last phenomenon is responsible for the common designation "sickening" which is applied to these but never to cutaneous pain. This sickening pain is not closely localized and while, of course, due to stimulation of afferent nerves the sensation is not comparable to that associated with the picking out of pain spots on the skin. Like visceral pain also, it is frequently referred to some area distant from the lesion so that acute fibrositis is often diagnosed as thoracic or abdominal visceral disease, angina, pleurisy or gall-bladder disease if it originates in the thoracic region of the back or migraine or sinus headache if the lesion is in the neck or scalp.

After the acute attack has subsided the nodules often remain permanently as tough, well organized fibrous nodules which are no longer tender. These give definite evidence of a previous attack and the person who has them is more liable than the average to develop a fresh acute attack of fibrositis which will produce new nodules and not affect the old ones. In the sub-acute or chronic cases, the onset may be gradual and insidious or it may be a result of several acute attacks after which the definite nodules become palpable.

In a large proportion of patients with sensitive nodules there is another condition which is equally definite on physical examination but which is not susceptible to pathological examination. This is the condition of muscle spasm. It is found most frequently in the back of the neck or in the lateral spinal muscles of the thoracic region and gives the feeling of a smooth, firm, painful area of muscle extending from two to four vertebræ or in some cases almost half the length of the spine. The remarkable feature is the ease with which it may be made to disappear when the joints of the spine are hyperextended. Both the local tenderness and the referred pain may be made to vanish, much to the patient's surprise, by a simple manipulation which will be described

later. This rapid change suggests that the underlying process is a functional contraction of the muscle rather than a pathological process which could be demonstrated by microscopic study. One patient who was in the terminal stages of subacute bacterial endocarditis developed such extreme pain in the lumbar muscles with intense spasm that no ordinary measures would give any relief. Under an anæsthetic he was moved on to a frame which gave hyperextension of the spine. After one day on this there was considerable relief. When he died about three days later, from a cerebral embolus, the whole of the lumbar area was examined most carefully and sections of bone and muscle showed no abnormality.

Fibrositic nodules and areas of muscle spasm may both be associated with osteo-arthritis in older patients. Until treatment has been carried out for some time it is difficult to make sure that the symptoms are due to fibrositis. Even in cases where there is x-ray evidence of a degree of osteo-arthritis of the spine, the patient should get the benefit of treatment but more care will have to be taken where there is extensive bony change, to avoid force in any of the manipulations. Where osteo-arthritis is quite extensive, one is inclined to feel that the referred pain is due to it and that little can be done to give relief, but care should be taken always before making this decision. It is not rare to find very marked x-ray evidence of hypertrophic bone change in the spine with no pain and no history of previous pain. This finding also does not prove that there is lack of normal mobility and indeed it is found in those who do strenuous physical work or take part in sports which demand at least normal flexibility. If the typical tender nodules are also present, it is well worth while having them treated, for in many cases the patients have been helped greatly even though there could be no change in the condition of the joints.

A few examples may be given of the typical complaints and the results of treatment.

Headache is commonly associated with tender nodules in the muscles at the back of the neck. It is most often occipital in distribution but may extend forward to the forehead where it will usually be unilateral. It is sometimes present all day or for several days at a time and is not brought on by reading or other use of the eyes. In most cases the patient will also complain of tension in the neck muscles. A common feature of this headache is that it is present on waking in the morning and eases during the day; it is also worse if one sleeps in later than usual, so a Sunday morning headache should suggest this as the cause. In the susceptible patient fatigue or worry will often give tension in the neck with headache. The fibrositis and muscle spasm of the neck region may also give symptoms referred to a branch of the brachial plexus. Of this one example is a case of neuralgia of the ulnar nerve area with paræsthesia of the corresponding fingers. The symptoms were present for over two months before treatment was directed to the neck. They disappeared in less than two weeks and did not return.

The patient who suffers from migraine may have neck muscle tension as the cause of greatly increased frequency of the typical migraine attacks. With suitable treatment there is usually a change in a few days from daily sickening headaches to relative freedom from distress and later there will be only a rare attack of the underlying migraine, usually associated with fatigue or worry.

Above the scapulæ the trapezius muscles are often tender and painful but pain is not often referred from this area except to a small spot in the upper arm near the insertion of the deltoid muscle.

From the region of the thoracic spine referred pain is fairly common and in many cases there is no complaint of pain in the back. The area of reference may be nearly anywhere in the front of the chest or abdomen; it is usually three or four inches to either side of the midline and therefore the patient may think of heart disease or gall-bladder disease. Palpation will demonstrate muscle spasm at the corresponding area in the back and hyperextension of the spine will often give most remarkable relief. A woman of sixty who has hypertension recently complained of steady pain in the left chest over an area from the second to the fifth interspace and from the edge of the sternum to the axilla. Her physician had advised bed rest which she continued for three weeks with no real relief. On examination there were definitely tender areas over the intercostal nerves in front and there was unilateral muscle spasm to the left of the spine. Extension of the spine gave nearly complete relief at once

and the rest of the pain disappeared in a few days more with exercises at home.

Not infrequently an extremely tender area may develop along the origin of the deltoid muscle about two-thirds way out on the inferior border of the spine of the scapula. Pain may be referred from it to the outer side of the arm in the area supplied by the fifth and sixth cervical nerves but there is seldom any complaint of pain at the scapula itself.

METHOD OF EXAMINATION

In the clinical examination it is important to get the patient's muscles well relaxed. Then a routine of palpation may be followed starting in the neck close to the skull and just behind the mastoid processes. From there the area to be examined extends around behind to the corresponding point on the other side. Palpation should be quite firm and across the length of the muscles. In this area the transverse processes of the upper cervical vertebræ are always tender on firm pressure and should not be mistaken for fibrositic nodules. Following downward the palpation continues, never going farther forward than the line from the mastoid process to the top of the shoulder. One may palpate the muscles above the upper edge of the scapula most easily by standing behind the patient who is seated on a stool; quite firm pressure must be used, and again the direction of the movement is across the line of the muscle fibres. In the palpation of any of these areas the fingers should be placed firmly on the skin and then moved over the deeper structures without sliding on the skin. If this method does not give enough relaxation, the neck and shoulders may be examined by having the patient lie flat on his back without a pillow while search is made with the finger tips for tender nodules.

To examine the thoracic region the patient should be lying prone; the area to be examined lies on either side of the spines for a distance of two or three inches, that is the area between the two scapulas. It is not common to find the nodules much below this region until one comes to the crest of the ilium. Then there are often some of them just below the crest and along the line of the sacro-iliac joint.

The symptoms of fibrositis are of such widespread distribution that it is always advisable to include a search for tender nodules as a routine part of every physical examination. When this palpation is carried out as a routine, nodules are found in about one-quarter of all patients. This does not mean that all these people have pain from the nodules or even that the spots are tender to pressure; however it is true that they have at some time or other had an attack of fibrositis. Also those who have nodules are susceptible to further attacks under conditions of strain and fatigue. In contrast to this, areas of muscle spasm are always tender and may be associated with local or referred pain.

TREATMENT

In a general way treatment may be said to be entirely in the realm of physiotherapy. These patients get marked relief by treatment suited to the severity of their condition. In the extremely acute case with sudden onset, heat and rest with the use of analgesic drugs may be necessary for the first few days before massage can be used. In the more chronic cases manipulation and deep massage can be used from the start. The fault most often found in massage treatment is that it is too gentle to produce results. However, this does not mean that the patient's further treatment should be taken over entirely by a massage department; the medical practitioner can do much and often the patient under his direction can continue the treatment and bring it to a satisfactory conclusion. The essential part of treatment is to stretch the tissues involved, by some means or other, either by traction where that is possible or by firm massage directly on the tender nodules. Some authors have advised the injection of a local anæsthetic solution directly into the nodules with the object of breaking them up by the pressure of the fluid. If the nodules are fairly large it is possible to do this but it is no more effective than firm massage; if the nodules are quite small and deep in a muscle it is almost impossible to inject the solution accurately. It will be noted that the rest of this section on treatment is devoted mainly to manipulative methods and exercises which are directed to the relief of muscle tension and spasm. In most cases, as previously indicated, the nodules and muscle spasm are present in each patient in varying degrees and therefore both massage and exercises are usually needed to effect a cure.

Before any manipulative treatment is undertaken it is wise to have x-ray films made of

the spine to rule out abnormal bone conditions and to be sure that the bones are not partially decalcified through old age, and therefore fragile.

The treatment of the neck and shoulder areas, when they are involved, should be done first. The table or couch should be firm but not hard and the head end of it should not be raised. The patient is to lie flat on his back with the shoulders at the head end of the table and the head supported by the physician's left hand. The right hand is placed below the patient's chin and direct traction exerted (Fig. 1). This should not be more forcible than is necessary to relax the neck muscles and there should be no suggestion of moving the patient's trunk by the pull. This steady traction should be continued for about one minute and may be repeated at the end of the other manipulations. Next the head is held firmly by a hand on each side and moved upward and toward the patient's feet while at the same time the face remains looking directly upward (Fig. 2). This manœuvre is easy to do but difficult to describe and it is a movement which can not be done voluntarily. Next the head is held firmly again and moved laterally, always being kept with the line from forehead to chin parallel to the length of the body (Fig. 3). This is not to be confused with the ordinary side bending of the neck. The next thing is to rotate the head toward each shoulder (Fig. 4). At the extreme limit of movement the head is held firmly for about fifteen seconds to stretch the tense muscles and fibrous tissue. Finally direct traction is employed as at the start. Each of these movements of the head and neck should be done deliberately, without hurry and with a firm grasp of the head to give the patient confidence and to promote maximum relaxation; in no case should so much force be used as to give actual pain.

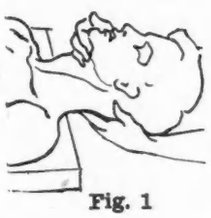


Fig. 1

Fig. 2a

Fig. 2b

Fig. 3a

Fig. 3b

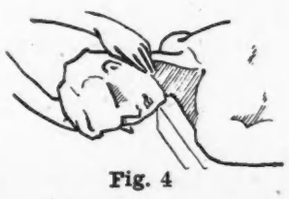


Fig. 4

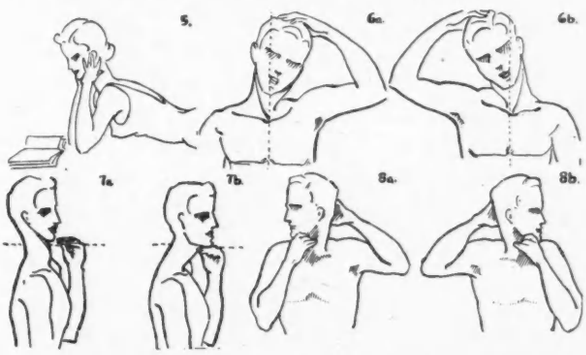
After the manipulation the patient should sit up and be given the following instructions about general care, and special exercises which he can carry out until seen again.

1. When sitting, sit in a fairly straight chair with the hips well back in the chair and avoid sitting with the legs crossed. (The latter direction applies mostly where the lower part of the back is involved.)

2. When in bed do not prop up with pillows to read. There is no position that gives pain in the neck and shoulders more quickly than lying with the head forced forward.

3. Without restricting clothes lie prone on the floor daily with the hands supporting the chin and the elbows on the floor about twelve to eighteen inches apart. This is the posture so often adopted by children to read. By staying for five minutes in this position the muscles relax and the thoracic spine becomes extended. To increase the pull on the back the elbows may be placed a few inches farther forward (Fig. 5).

The special instruction for daily treatment of the neck region are as follows: (the exercises are to be done while sitting straight.)



1. Place one hand on top of the head and draw the head toward that shoulder (Fig. 6a and 6b). Normally there is no discomfort in doing this but the person with fibrositis finds that the movement produces pain on the opposite side of the neck. This pull should be held so as to stretch the tender muscles for about fifteen seconds. Then a similar movement is done to the opposite side.

2. Bend the head forward with the chin nearly touching the chest and, while keeping it down, bend it to each side. The hand is placed on the top of the head again and forward pressure exerted to stretch the muscles at the back of the neck and the chest.

3. While sitting upright move the head directly forward without lowering the chin. This gives the effect of sliding the chin forward on a straight line. Then draw the head back as far as possible, still keeping it in the same horizontal line (Fig. 7a and 7b). This

exercise is done without help from the hands and should be repeated several times.

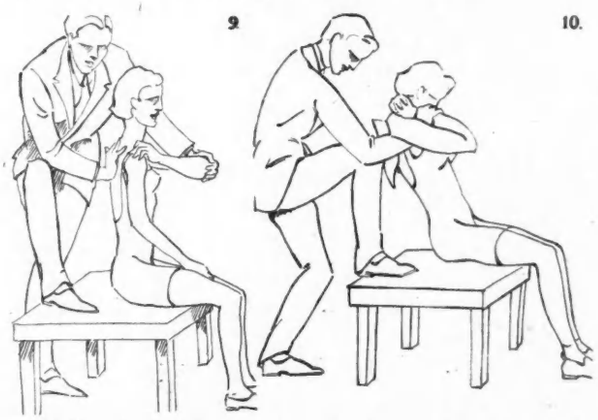
4. Rotation of the head toward each side. This is to be done by looking to the right, then the left hand is put behind the head to assist in pulling it around and the right hand grasps the chin to push it further. On looking to the left the position of the hands is reversed (Fig. 8a and 8b).

If the main lesions are in the trapezius muscles, some one in the patient's family may be instructed in the simple type of massage which is effective. The patient should sit on a stool and the massage of the nodules is conducted from behind. It may be done with the finger tips which should be placed firmly on the skin. The skin is then moved over the underlying tissues in a direction across the line of the muscle fibres or in a rotary manner. When the tender nodules are very firm, painful pressure should be exerted on them. At the beginning of treatment the degree of pressure may best be gauged by the results next day. If there is no change the pressure should be firmer. If the pain and tenderness are more marked the force has been too great. If there is some improvement treatment should be continued with the same amount of pressure. This massage is to be done for about five minutes daily. If the nodules are on either side of the spine the patient should lie face downward on a narrow cot so that the arms can hang over the sides and thus draw the scapulæ apart to give more space for massage.

When the lesion to be treated is spasm of the muscles on either side of the thoracic spine the method of treatment is somewhat different. The part to be done by the physician is extension of the spine with the patient sitting on a stool. This may be done in a number of different ways of which two will be described.

The first method is to have the patient place his left hand on his right shoulder with the elbow directed forward. This elbow is then grasped firmly by the left hand of the physician who is standing behind him and then a firm, quick push is given by the right hand to the affected segment of the spine. This pressure is repeated several times and may be applied in succession to a number of different areas (Fig. 9).

The second method is effective mainly where the spasm is in the upper half of the spine. The patient sits on a stool, puts his hands behind his neck and locks the fingers together. The physician places his right foot on the stool behind the patient and places a small pillow against the patient's back to guard it from bony pressure of the knee. Then the doctor's arms are stretched forward, palms of hands downward, under the patient's arms and the latter's wrists are grasped firmly. This gives a very secure hold which allows one to lift the patient slightly and at the same time draw him back against the pillow so that the knee exerts sufficient pressure to extend the joints of the spine (Fig. 10). During this manœuvre, if sufficient relaxation is obtained, it is quite common to get a cracking noise which is exactly the same in character as that obtained by pulling on one's fingers. The noise is probably produced by the sudden tension of a joint capsule, as the soft intervertebral disc changes shape with increased pressure on its posterior margin and decreased pressure anteriorly. It is not necessary to produce this noise in order to do good but when it happens one can be sure that good relaxation was obtained. In elderly patients the bones are usually thin and partly decalcified. Therefore this latter manipulation should not be used and all forms of treatment should be carried out much more gently than in young healthy adults.



Following this treatment the patient is given directions for an exercise to be done at home before going to bed. He will make a firm roll of cloth from a towel or small sheet, about two inches in diameter and tie it with string to keep it rolled. This is placed on the floor and he lies down with the roll at right angles to the spine and crossing about at the tenth rib. Then he moves his arms so that the hands touch the floor as far as possible beyond his head. The hands are kept touching the floor in this position for about thirty seconds and are then brought down to move the roll about two inches further up the back. The arms are then extended as before for the same length of time.

This is continued until the roll has been moved up about as far as the fourth or fifth thoracic spine and this concludes the exercise. A person who has normal flexibility in the back can do it with no discomfort; those who have marked tension may not be able to carry out the whole exercise for the first few weeks.

Most patients who will persist with these various exercises at home will get improvement. They should return at intervals for the manipulations in the office and if it is then found that the tenderness is not clearing at a satisfactory rate, professional massage should be obtained.

SUMMARY

1. Fibrositis is one of the commonest causes of backache and of pains referred from the back.

2. Along with fibrositis, in most cases, there is also muscle spasm in a limited area.

3. The pathology of these conditions is described in detail and it is noted that they are not inflammatory in character.

4. Methods of treatment are described which consist of manipulations to be done by the physician and of exercises to be done by the patient.

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MARCH HÆMOGLOBINURIA*

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MARCH hæmoglobinuria is the term applied to the condition in which free hæmoglobin appears in the urine during and following erect exercise, as running and marching. This hæmoglobinuria is preceded by an elevation above normal of the free hæmoglobin in the blood.

Fleischer[1] in 1881, was the first to report the condition. Prior to the first Great War the majority of cases had been reported by Germans. Dickinson[2] described a case in 1894 and Finny[3] another in 1926. Watson and Fisher[4] reported their case in 1935. These three were the first appearing in the English language. Gilligan and Blumgart[5] studied three cases in 1940. They reviewed all previous cases, numbering 40, and did extensive work on the quantitative aspects of hæmolysis. Several cases have been reported by Allied Army medical officers since 1940. Among them were two in the Canadian Army Overseas, observed by Palmer and Mitchell[6] whose studies in quantitative analyses, published 1943, confirmed the results obtained by Gilligan and Blumgart.

All reported cases of march hæmoglobinuria have occurred in males; the initial attack appearing between the ages of 16 and 35. The paroxysms may occur over a period of three to six months and then completely disappear. Occasionally they may be evident for several years. Exercise in the erect position is a necessary factor in the production of the paroxysms. Several of the cases reported have occurred in marching soldiers, hence the term "march hæmoglobinuria". Athletes during training runs and when participating in such games as football have developed the syndrome. Exercise in other than the erect position does not produce paroxysms, *e.g.*, cycling. Following erect exercise the patient notices that his urine is red in colour, occasionally almost black. This colour usually disappears in three or four hours. Exceptional cases may require twenty hours or longer for the urine to return to its normal colour. The red urine gives a positive benzidine reaction, and contains very few or no red blood cells. Spectroscopically, oxyhæmoglobin is the pigment causing the red colour. At the height of the paroxysm the blood plasma is tinged pink to red. It is clear and the spectroscope shows the typical bands of oxyhæmoglobin. No chills or fever accompany the paroxysm but vague abdominal distress, pains in the back and thighs may be present. Often hæmoglobinuria is the only symptom.

Physical examination reveals no constant abnormality which may have a predisposing effect in causing the hæmoglobinuric episodes. Certain cases have a slight lordosis which has been thought to be a factor. At rest, all laboratory findings are within normal limits, except that traces of albumin may be found in the urine, suggestive of orthostatic or other albuminuria. Anæmia is not a characteristic of this syndrome. (In certain other types of paroxysmal hæmoglobinuria, anæmia is a prominent symptom). Red cell fragility as determined by the hypertonic saline method does not vary from normal and no evidence of fragility is demonstrable by

*The case to be reported was under observation while training in Camp Ipperwash during March, April and May, 1945.

any other method. This obtains during rest and at the height of a paroxysm. Chilling, local or general, does not precipitate an attaek. The Donath-Landsteiner reaction is negative. As yet, no auto-hæmolysin has been demonstrated in the blood of these patients.

CASE REPORT

Pte. J.F.C., aged 18 years 3 months. The family history showed nothing significant. Before enlistment he engaged in various strenuous sports and the training activities of a reserve unit, including marches, without untoward effect. He enlisted January 2, 1945, when his urinalysis was recorded as negative for sugar and albumin. He first noticed that his urine was dark-coloured near the end of February following a march, and again three weeks later, when he consulted the medical officer. Examination of his urine showed no red blood cells but gave a positive benzidine reaction and was positive for albumin. He complained of some abdominal distress and nausea, stating that his "stomach felt tightened up" for four or five hours each time.

Examination showed him to be bright and alert although somewhat immature physically. His height was 5′ 5″ and his weight 123 pounds. There was no evidence of jaundice. The superficial lymph glands were normal. His tonsils were moderately enlarged but free of evidence of infection. The heart and lungs were normal. His spleen, liver and kidneys were normal in size without any tenderness. All reflexes were normal and there was no evidence of luetic infection. He had a moderate left varicocele. The blood pressure was 110/75. His resting urinalysis on several occasions was normal with the exception of an occasional trace of albumin. A Mosenthal test was negative for imperfect kidney function. The Wassermann reaction was negative. His red cells numbered 4,990,000 and the white cells 6,800. The differential white count was within normal limits and the sedimentation rate was 3 mm. in one hour.

Escorted by an N.C.O., this man was sent on a controlled march of approximately six miles. His urine was examined at the end of three miles, after six miles and at two-hour intervals thereafter until it was normal. He wore the usual training equipment which included rifle, respirator, steel helmet and haversack. Below is recorded urinalyses during and following the march.

TABLE I.

Time	*Appearance*	*Alb.*	*Hb.*	*R.B.C.*	*Casts*
Before march..	Clear, straw coloured	Trace	Nil	Occ.	Nil
After 3 miles...	Very dark red	2 +	Pos.	Occ.	Numerous granular
After 6 miles...	Very dark red	2 +	Pos.	Occ.	Numerous granular
11.00 hrs..					
13.00 hrs..	Dark red	2 +	Pos.	Occ.	Few casts
15.00 hrs..	Pink	1 +	Pos.	Nil	Occ. cast
17.00 hrs..	Straw coloured	1 +	Neg.	Nil	Occ. cast
19.00 hrs..	Straw coloured	Nil	Neg.	Nil	Nil

It is noted that his pre-march urine contained a trace of albumin which increased during the paroxysm and did not disappear until after the complete disappearance of the hæmoglobinuria. Upper abdominal distress was present during the paroxysm. Blood taken by venipuncture following the march was allowed to clot. The supernatant plasma was clear and had a reddish colour. Examination of this boy's abdomen immediately following the march showed slight distension and tenderness in the epigastrium; otherwise his abdomen was normal.

The abdominal distress accompanying the hæmoglobinuria suggested a trial march while fasting. Accordingly on two consecutive mornings without breakfast he was sent on smart walks of thirty minutes, without equipment. He returned from these walks without gastric distress or hæmoglobinuria but his urine was positive for small amounts of albumin which disappeared in two hours or less. Three hours after eating his breakfast, the walks were repeated. He returned from these with the usual dark red urine and abdominal distress. His urine gave positive benzidine reactions and was negative for red blood cells. On other occasions, thirty minute walks seven and eight hours after breakfast and without lunch caused no symptoms other than a moderate albuminuria.

The table below gives average findings of urinalyses and symptoms during fasting rest, following fasting walks, and post-prandial walks, obtained from repeated trials.

TABLE II.

Urine specimen	*Albumin*	*Hæmo-globin*	*Abdominal distress*
Fasting rest.....	Nil to trace	Nil	Nil
Fasting walks 30 minutes.....	Trace to 2 +	Nil	Nil
30 minute walk..... 3 hours after meal....	2-3 +	Pos.	Pos.

Subjecting this boy's feet and lower legs to ice water for twenty minutes did not cause a paroxysm, and an hour's stiff cycling was equally ineffectual.

Fluoroscopic and film studies made of the upper digestive tract while fasting and at the height of a paroxysm were entirely negative for any abnormality. It was thought that some factor might be demonstrated which possibly would explain his abdominal distress, such as involuntary muscle spasm.

A march following a fast broken by the ingestion of twenty-four ounces of barium failed to precipitate a paroxysm of hæmoglobinuria, but was attended by a certain amount of gastric distress. This experiment may be disregarded, since two forty minute walks after meals the next day showed that he was entering a remission. No hæmoglobin was excreted but he suffered mild abdominal distress. Within four days he was able to complete smart walks lasting 30 minutes or longer without the appearance of any of his erstwhile symptoms. It is interesting to note that on entering his remission the hæmoglobinuria disappeared first, to be followed in a very few days by the abdominal distress and the albuminuria. Since entering his remission, several urinalyses of his resting urine were entirely negative for any trace of albumin.

DIFFERENTIAL DIAGNOSIS

Fresh urine which is pink, red or black may owe its colour to hæmaturia, hæmoglobinuria or to some pigment-producing substance which has been ingested, such as beets or phenolphthalein. A positive benzidine reaction indicates the presence of either whole blood or free hæmoglobin. Myoglobin, from muscle tissue, very rarely present in urine, also produces a positive reaction. Myoglobin and oxyhæmoglobin are determined spectroscopically. Hæmoglobinuria may occur in association with severe acute infections.

Malaria in a virulent form seems to be necessary in the development of blackwater fever, which has hæmoglobinuria as one of its symptoms. Hæmoglobin may appear in the urine following the ingestion of various poisonous substances.

March hæmoglobinuria is included in a group of syndromes which have an irregularly occurring hæmoglobinuria. These are known as paroxysmal hæmoglobinurias and are briefly described below. A minute description of the first three was given by Witts[7] in 1936. Luisada[8] in 1941 published a full account of the fourth.

Paralytic hæmoglobinuria. — This disease is characterized by red urine containing myoglobin and a rapidly progressing paralysis of the muscles. It is frequently observed in horses and has been reported in man, although extremely rare. It occurs in well fed work horses following a two or three days' rest. During the rest much glycogen is stored in the muscles. On resumption of work, excessive amounts of lactic acid are freed from the glycogen. The lactic acid damages the muscles, thus liberating myoglobin which is excreted by the kidneys.

Syphilitic type or hæmoglobinuria e. Frigore. —The hæmoglobinuria in this syndrome is induced by exposure to cold. Chilling of the hands and feet in ice water for ten minutes is sufficient. Patients subject to this type of hæmoglobinuric paroxysm have positive Wassermann reactions or clinical evidence of syphilis. The Donath-Landsteiner reaction is positive, indicating the presence in the blood of a specific hæmolysin which is activated when the patient is exposed to local or general chilling. In all other types of paroxysmal hæmoglobinuria this reaction is negative. Hæmolysis does not persist between attacks. Adequate anti-syphilitic treatment disposes of this particular syndrome.

Marchiafava-Micheli type of hæmoglobinuria. —There is no known cause for this disease, nor is there any treatment. Neither syphilis nor malaria enter into its etiology. It begins insidiously, with gradually developing anæmia and many years may pass before the paroxysms of hæmoglobinuria appear. The later stages of this pathological entity are characterized by fully developed hæmolytic anæmia and paroxysms of hæmoglobinuria leading to death in three to five years. The paroxysms are likely to appear during sleep at night. They do not occur during sleep in the daytime. Changes in posture, exertion, cooling, atropihe or pilocarpine have no effect in their production. The Wassermann and Donath-Landsteiner reactions are negative. Aside from the paroxysms, the symptoms of this disease are due to severe anæmia.

Favism.—This is an ancient disease in the Mediterranean area, especially in Sardinia. It appears less frequently in Sicily, Italy, the Greek Islands and North Africa. The disease is due to the ingestion of the seeds or the inhalation of the poisonous principle of the plant *vicia fava,* commonly known as the broad- or horse-bean. Symptoms develop within a few minutes following inhalation or within a few hours following ingestion. General malaise, headaches, dizziness and nausea appear early, later followed by chilling, lumbar pains and fever. Still later hæmoglobinuria and jaundice occur. The jaundice may be very intense. Fatal cases die within the first two days. In those who recover, the hæmoglobinuria and fever terminate in five or six days. Children nearly always die. The hæmoglobinuria may last three days, if longer the patient generally dies. There is no treatment except abstention from contact with the plant *vicia fava.*

DISCUSSION

This case exhibited the syndrome of march hæmoglobinuria for almost three months and was under observation during the last two. Time and facilities for detailed laboratory studies were not readily available, hence further investigation could not be done. However, the history, negative Wassermann and inability to produce a paroxysm when exposed to chilling, as well as the remission, rule out any other type of paroxysmal hæmoglobinuria.

It was observed in this case that the presence of food in the upper digestive tract, as long as three hours after its ingestion, was a factor in the development of the paroxysms. When the upper digestive tract was empty, that is seven or more hours following a meal, erect exercise did not precipitate any symptoms. Therefore, in this patient, either the mechanical presence of food in the upper digestive tract, or the process of its digestion and assimilation was necessary in the production of the symptoms of march hæmoglobinuria. His remission prevented further investigation. It was intended to determine whether his symptoms appeared after the ingestion of an inert meal such as a barium chloride mixture, when presumably only the mechanical factor would be involved. If not, then the effect of fat-free, protein-free

and carbohydrate-free meals were to have been determined, or meals composed entirely of any of the three.

During the period of observation it was also noted that generally, this case's fasting and resting urine showed no albumin, but frequently a trace when not resting. Erect exercise caused the appearance or the increase of albuminuria at all times, but only after meals did hæmoglobin appear in the urine. The hæmoglobin always disappeared from the urine before the complete disappearance of the albumin. Finny[3] noted this continuing albuminuria in his case, as also did Gilligan and Blumgart[5] in theirs. Fisher and Bernstein[9] noted that their case developed 3 plus albuminuria, without hæmoglobinuria, following a walk.

Gilligan and Blumgart[5] found that the amounts of whole blood hæmolyzed in any one episode were only between 6 and 40 c.c., and that less than 15% was excreted by the kidneys. Palmer and Mitchell[6] obtained similar results.

Gilligan, Altschule and Katersky[10] investigated the excretion of injected hæmoglobin in normal subjects, in a case of march hæmoglobinuria, and in cases of pre-existing albuminuria. Varying amounts of stroma-free solutions of hæmoglobin were injected intravenously. They found that in normal individuals no hæmoglobin was excreted under plasma concentrations of 135 mgm. %. In plasma concentrations above this, hæmoglobin was excreted and the excretion continued until the plasma level reached 40 to 50 mgm. %. These findings are similar to those of Ottenburg and Fox.[11] In those cases of pre-existing albuminuria, as in chronic passive congestion of the heart, hæmoglobin appeared in the urine at plasma levels of 40 to 50 mgm. %. In the case of march hæmoglobinuria, injected hæmoglobin was excreted at the same low level of approximately 40 mgm. %, as occurred when he suffered a paroxysm. Always less than 10% of the total injected hæmoglobin was excreted. Normal subjects who received insufficient amounts of injected hæmoglobin to produce hæmoglobinuria did not develop albuminuria. Those whose injected hæmoglobin was sufficient to cause hæmoglobinuria, showed a proteinuria greater than the protein of the hæmoglobin excreted, and the proteinuria persisted for about an hour after the kidneys ceased excreting hæmoglobin.

The presence of albuminuria in many of the cases of march hæmoglobinuria reported, was not noted. Some observers remarked the association of albuminuria with the hæmoglobinuria, and some further observed its continuance after the hæmoglobinuria disappeared from the urine. These facts and the findings of Gilligan, Altschule and Katersky[10] in their investigation of the excretion of injected hæmoglobin, would indicate that there is some association between the albuminuria and hæmoglobinuria, if not in the actual hæmolysis, at least in the excretion of hæmoglobin at below normal thresholds. The excretion of hæmoglobin in march hæmoglobinuria may represent a further stage in the condition which causes the albuminuria in some of the so-called functional albuminurias.

No reasonable theory can be formulated to explain why an apparently normal young man should suddenly exhibit for three months hæmoglobinæmia, hæmoglobinuria, albuminuria and abdominal distress after walking for thirty minutes. Nor can any explanation account for the fact, that for at least part of the three months, the appearance of the above symptoms depended on the presence of food in the upper digestive tract.

SUMMARY

A case of march hæmoglobinuria is reported.

Observations were made in this case which indicated that the presence of food in the upper digestive tract was a necessary factor in the production of a paroxysm.

The association of albuminuria with the hæmoglobinuria is briefly discussed.

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SURGERY OF THE SYMPATHETIC SYSTEM*

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Ad augusta per ardua: V. Hugo, *Hernani*, 4th act (password of the conspirators).

NEURO-SURGERY has long confirmed the fact that the removal of the frontal lobes or the resection of a whole cerebral hemisphere for a voluminous tumour of the brain does not produce an appreciable change in the intellectual equipment of a patient. This highly specialized surgery has even, in some cases, increased the mental acuity.

There has been a long controversy to decide if the brain anatomist can with his scalpel demonstrate the reasons for genius or insanity. Franz Gall, in spite of his empiric and absurd theory of phrenology directed scientific minds towards the autopsy of the cerebral nervous system in cases of certain intellectuals, who bequeathed their brains to the scientists for analysis. In 1860, Rudolph Wagner compared the encephalons of three men of genius with that of an ordinary day labourer. A very close examination of the convolutions, their depth, their number and their pattern, their respective weights, showed no dissimilarity. Since then it is not certain that a highly endowed persons has more intricate gyri than those of a moron.

A little later, a special study of the frontal zone, where the intelligence centre was supposed to be located, did not elicit a marked unlikeness between the frontal lobes of world-wide known men and those of average intelligent men.

Brain students did not find the physical substratum of cerebration because they had always turned their attention towards dead brains until they began to consider the driving power of the cerebral matter, the fuel, so to speak, that kindled the thought, in this instance the blood which nourishes the cerebrum.

The most serious mistake of former dissectors was to examine the organ after they had removed and discarded the meningeal coating which holds the blood-supplying net-work. The study of the volume and complexity of these vessels gives more information concerning the cerebral power than the weight, the size and the intricacy of the fundamental structures themselves.

In 1926, Hindsie noticed a greater richness in the composite aspect of the vascular contribution in the meninges of intellectually well-equipped people since the membranes that cover the brain of men of genius contain large numbers of over-sized diameter vessels. On the other hand, the half-wit or the ninny is provided with tiny arterial coverings which, furthermore, have a narrow calibre. Moreover, the chemical composition of the blood plays a very important rôle in fostering intelligence. The amount of sugar, of calcium and other main elements assume great significance.

Salomon Katzenelbogen and Harry Goldsmith have minutely inquired into the dosage of the blood calcium in the various types of mental diseases and they have brought to light the interesting fact that most of the cases of insanity of organic origin have a smaller lime content than normal persons. Calcium, however, accounts for only one factor. Katzenelbogen and Freidman-Buchman have revised the quota of sugar in several instances of mental trouble which they found to exceed the usual limits. This truth is especially plain in the split-personality insanity. The higher the cerebro-galactose, the higher the nervous tension of the individual. That the abnormally sugar impregnated brain belongs to an insane individual is conclusive.

Though we cannot, at the present time, better the mental power by altering the chemical constitution of the blood, William Healy, William Sargant and J. M. Blackburn have substantiated that benzedrine can quicken mental responses. They have, in two groups of patients, proved that the drug absorbers were mentally superior by 8.7% than the controls.

The experiments of the German Fleischle von Marxow, in 1890, to detect minute electrical currents through the skull of different animals were resumed by Hans Berger, of the University of Iena and by some American scientists. This led to the discovery of the alpha and beta rhythm cerebral waves. Donald B. Lindsay's researches of the Brush foundation and at the Western Reserve University showed that the alpha waves appear in three year old children to increase in frequency and amplitude up to 8 or 10 years where they reach the adult level. He believes that the brain electrical energy grows during adolescence and that it may be

* Read before the Montreal Neurological Society, at the Montreal Neurological Institute, February 17, 1943.

related to the physiological mutations of this stage of life.

Amongst other workers, H. H. Jasper and H. L. Andrews after having succeeded in eliminating the organic processes which affect the observed potential at the surface of the skull at the touch of the electrodes, have fully demonstrated, after many attempts, the autonomy of the various generating-wave regions out of the proper manipulations of these electrodes over all the cerebral fields. They suggest that the alpha frequency might be controlled by the cerebral basal ganglia. They have also established, by operating upon limited territories, either occipital or Rolandic, that there was no correlation between the occipital alpha waves and the beta Rolandic zones.

According to their remarkable experiments Jasper and Andrews have clearly proved that the alpha and beta waves rightly originated from the cerebrum and that the electro-encephalogram is apt to take its place amongst the diagnostic ways of the encephalopathies, mainly the tumours and the epilepsies.

In regard to these researches it is a little baffling and stinging to state that a woman thinks faster than a man. The average frequency of the alpha waves is 11 per second in a woman against 10.2 per second in men, hence the deduction that the feminine sex is a faster thinking one. The cerebral waves are as individual as the finger prints, to such an extent that Professor Lee, from the Iowa State University looks at the possibility of filing the cerebral waves just as the finger prints are taken.

The picture of the intelligence becomes neater and neater. The brain students are now sure that intelligence does not depend, in all cases, upon the size or the weight of the cerebral matter, as was heretofore believed, but upon the amount and quality of the blood and the way in which this blood is influenced by the internal chemical transformations which can also effect the electrical energy of the brain.

GENERAL CONSIDERATIONS

In accordance with the above mentioned facts all the experiments essayed on the cerebral system must end in the blood circulation, the paramount factor in the regulation of all parts of the body. The function and the nervous equipment have been worked upon; the vascularization has been discussed and described; the pathological anatomy of the tissues has been exhaustively examined. Cerebral arteritis has been long known. But are the effects of vasospasm often mentioned? If one will refer to the different articles I have published, since 1931, in several medical reviews, one will remember my insistence upon the origin and the mechanism of the vaso-motor troubles. I do not try to reduce everything to a single formula, that is, vaso-spastic phenomena, but I think that this part of the question has not been enough scrutinized. It is vaso-spasm that rules the evolution and the prognosis in thrombo-angiitis obliterans; in senile or senilo-diabetic arteritis; in physiopathic troubles; in digestive tract disturbances; in cardio-pulmonary system disorders, in the pathological manifestations of the cerebro-spinal system.

In fact, in organic arteritis, so frequent, though concealed or secretly progressing, the vessel may be touched by some discrete sclerotic plate or by a slight parietal change (Monckeberg type), sufficient nevertheless to turn loose series of reflexes disjointing the blood irrigation mechanism. The pain reported by the patient concerns the inmost order of the peri-arterial plexuses strangulation, by vaso-constriction, which, not identified, brand the patients with a neurotic stigma. The vascular spasticity will lessen the blood irrigation of the tissues. The humoral condition suffers from this restriction, it is disturbed, the exchanges go wrong, the vital oxygen is inadequately distributed, the texture is blocked up with carbonic acid, waste accumulates in the interstitial zones, obstruction sets in, saline concentration decreases, function is gradually impaired, injuries happen in the iner part of the tissues, a lesion settles which afterwards automatically develops.

On several occasions I have happened to be an immediate witness of cerebral troubles or to be consulted for that reason. The patients complained of intense headaches whilst a mono- or hemi-paresis stood out which disappeared after a few hours or a few days without any sequelæ. Conversely, other cases presented fixed pathological phenomena.

In such a friable tissue as the cerebral matter, the least clash in the steadiness between the blood wave and the parietal contraction will disturb the distribution of the nutritious elements of the cerebral substance, its functions will be curtailed, its electrical energy will be reduced; calcium, sugar, phosphorus and other

vivifying principles of the regional life machinery will provide a more or less limited supply, therefore, ischæmia will settle, in a more or less large territory, the cells will become anæmic and devitalized, which conditions will bring on a cellular disintegration with hesitation, claudication, intermissions, fluctuations, locking or stopping of the regulation of the nervous influx, leading principles of the cerebral functions. Chaos, dislocation, anarchy in the cerebral life ordering is most often caused by arteritis with vaso-spasm as its inevitable corollary. If spasticity seems to be the only cause, it will ordinarily be of short duration but the after-effect should not be too much prolonged, otherwise there will appear irretrievable damage because of the cell frailty. When arteritis is in a more advanced stage, there will be a break in the arterial wall with a consecutive hæmorrhage the clot of which will put pressure upon the cerebral elements and will produce a surrounding attrition turning to the destruction of the affected area and crystallizing into a definitive hemiplegia. Reflexes will start from the broken point which will lead function astray through confusion and spasm, with a repercussion on the endocrine glands via the sympathetic system.

The cerebral vaso-constriction may still be instigated by the ductless glands, mainly the adrenals, whence hyperhormony is apt to influence the vasculo-cerebral system and to cause a more or less tight hypertonia according to the degree of the stimulus. After ovarian castration, the regulating curb of the arterial pressure gives way, hypertension may occur, repeated ram-like blows of the blood stream will end and produce a wear and tear parietal injury or reacting sclerosis to stand the blow, thence ideal grounds for vaso-spasm launching are constituted.

In hyperthyroidism, thyro-globulin transmuted into thyroxin, when its part of protein waste burning is done, will circulate in excess in the blood stream and, besides its elective fixation on the intra-intimal sympathetic endings of the vessels will assail the endothelium of the vasculo-cerebral network, to provoke erosions, priming of forthcoming arteritis and vaso-spasm as a result.

The hypophysis, pineal gland, retro-carotid corpuscle, gonads, have assuredly a system of hormonal elaboration the ordering and the impulses of which make themselves felt over the intra-mural sympathetic network, and so affect the cerebral substance.

The digestive tract toxins may also impair the cerebral arterial walls, may give rise in it to certain lesions the very likeness of which is sufficient to establish vaso-constriction.

The nervous stimuli, anxiety, worries, emotions, fears, apprehension, sorrows, overwork, connubial or private acrimony, failures or financial difficulties, moral or physical trials, induce disturbances in the travelling of the nervous influx which, striking the neuro-vegetative system or its annexes, bring forth troubles in nutrition.

MENTAL TROUBLES

The essential lines of mental unsteadiness are far from being always supported by organic lesions. A certain number of pathological disorders are made up of a disordered function, a rather vague definition which cloaks our ignorance. In my opinion these words have absolutely no meaning. Why is the working of one brain worse than that of another? The reason seems plain to me if we admit that the nutrition of the cells is ill secured. In fact, it is the want of balance between thoughts and action that makes mental aberration.

Why does the exteriorization of the mutations of the intelligence express itself in disorderly expression of the feelings, the affections, the customary dispositions? We know of a certain number of criteria to appraise the intellectual faculties, but how to weigh the unknown, amongst which is the imponderable, another elegant form of our scientific inadequateness? As for me, I am more and more coming to believe that the cerebral circulation must be concerned with the essential intellectual troubles and with their resulting manifestations. Convolutional atrophy is sometimes undoubtedly proved but this fact cannot account for everything.

Amyot states:

"Certain systematized delirious psychoses appearing to proceed from a mentally abnormal constitutional condition, such as the paranoid states, chronic hallucinatory psychosis, might be connected with delicate, degenerating progressing cerebral lesions, which can nevertheless tend to dementia. Unquestionable lesions of the nervous centres have not yet been found which could explain the pathogenesis of the maniaco-depressive disease. On the other hand, we know that melancholy is often a mental display of a senile evolution. Dementia præcox, including different aspects of psychological syndromes, is not perhaps an authentic disease; the term is used, anyway, to group these forms together and describes early developing mental troubles with an ominous prog-

nosis, with various facies but similar enough in different subjects. The cerebral lesions are histological and delicate ones; they harm the nervous cell and its dendrites, they are of the degenerating kind; they might also be inflammatory.''

As stated by Amyot, the anatomico-pathological substratum, except in unusual cases, does not correspond with the outward manifestations of the disorders. Post-mortem examination shows little and yet intellectual disturbances are there, threatening in their pathological consequences and their social counter-effects. The treatment of these irregularities will necessarily suffer from the unstableness of the propping point and will only be a symptomatic one. The therapeutic arsenal will consist of sedatives and narcotics, poorly worth ammunition to lead the fire against unknown positions.

CEREBRAL CIRCULATION

The internal carotid which gives off the middle cerebral or sylvian artery, the vertebral arteries which form the basilary stem, out of which spring the posterior cerebrals, the different communicating arteries, all the components of the Willis circle yield collaterals which bury themselves as terminal branches or which anastomose, travelling over the cerebral substance or sinking into it, securing tissue irrigation in all and every part of the brain, ruling the nutrition of all the cells and everywhere in the thickness of their walls, swaying the plexuses that regulate the vaso-motor condition. These plexuses attend the vascular network down to its furthest ramifications. Therefore vaso-spasm is bound to be felt in every fraction of the vasculo-encephalic system and thus to give rise to disorders, damage or cellular injuries bringing about disturbances or an annihilation of the function, if these irregularities chiefly graft themselves on a pre-existent arteritis.

Opposite the dysfunction of the cerebral life, a serious and important consideration must also be linked with the lesser or greater richness of the ramifications of the encephalon vascular network. To my mind, the distribution of the circulation must account for the intellectual defects in the demented patient. Hereditary disposition may play a part, although an intangible dogma cannot be set up on its component elements by reason of the reversibility occurring throughout the generations. Alimentary hygiene may interfere with the established deficiency in the organic cells if it is not adapted to the natural development of the individual. But I think it is my duty to insist upon the anatomical aspect of poor blood irrigation by an inadequate vascular latticework which will carry only a limited supply of the stocks necessary to the support, the normal re-adjustment and working of the cerebral life with respect to mental balance. It may be said that this explanation is imaginative. Maybe! But, what objection is there to speculation when supported by anatomo-pathological deductions?

With a needy or wretched arterial circulation, the cellular life of the brain is imperilled, altered, crippled. I say again that the constitutive elements of the cerebrum are rationed in raw and secondary materials, essential to their good working, whence degeneracy of the cell and consecutive debilitation of the intelligence, the manifestation of which will be disorderly and inordinate actions, the thought expressing of which will be confused, the exterior personality of which will be blurred, with morbific turn-taking impulsions, and at last, the characteristics of which will be melancholy, schizophrenia, dementia præcox, paranoid states, hallucinatory psychosis, in short, all the essential pathological mental troubles.

TREATMENT OF CEREBRAL VASO-NEURO-TROPHOSES

If the principle of the vasculo-cerebral system integrity is admitted, the cure of the lesions which may injure it seems easier to approach.

As I have just said at length, I have no doubt at all that by improving or by changing the brain vascular tonus, we will realize a transformation of the local nutrition and therefore, if the accidents are not irreversible, there will follow a more or less approximately normal restoration of the functions, inherent to the interested territory, either anatomical or intellectual.

Now, it happens that nature has set relays at the level of both right and left stellate ganglia, to which come and end all the vegetative fibres ruling the cerebral circulation, and which are contained in the carotid and vertebral nerves. These are exclusively made of gray rami communicantes, therefore form the peripheral offspring, that is to say, to terminate at the corresponding ganglia (stellate) without ever continuing their course as far as the cord. It is today admitted that there are no white rami communicantes above the first thoracic and

below the second lumbar segments of the cord. Only the protoneurons go through the anterior horn to scamper along the anterior root to make their synapses with the post-ganglionnic fibres, inside the sympathetic ganglia.

Consequently, if the stellate is the fibre cross-road of the cerebro-vascular tonus, it chances to be, because of its easy approach, surprisingly and properly located to be influenced by nupercain and the combination nupercain-alcohol para-vertebral injections. Such is the idea which guided us, Professor Léger and myself, when we decided to subject a certain class of patients *to the provoked cerebral vaso-dilation test.*

APPROACH TO THE STELLATE GANGLION

Several routes have been proposed for the para-vertebral injection of the cervico-thoracic ganglion. But of these I always use the posterior approach followed by Labat and White. Two needles are inserted in succession at a distance of 4 cm. from the midline, the first one between the 7th cervical and the 1st thoracic transverse processes, the second one between the 1st and 2nd rib, at the same distance from the corresponding spinous process. The needles should be stopped at a depth of 3 cm. below the ribs. Two c.c. of a 2:1,000 solution of nupercain are injected into the first needle which should bring on, in about five minutes, a Bernard-Horner syndrome and sympathetic signs in the hand, after which 2 c.c. of alcohol at 95° are added to consolidate the action of the nupercain. If the Bernard-Horner syndrome is not elicited, then I must contact the cervico-thoracic at the neck of the first rib through the needle introduced between the first and second rib.

APPRECIATION OF THE GENERAL END-RESULTS

If we analyze the results obtained in this series of cases, a first study eliminates three histories of patients who did not answer the questionnaire because of their having moved from their former address and who could not be retraced. Of the 37 observations, there were 14; seven of these were not improved and remained in their previous condition; 3 were temporarily ameliorated, 2 died after the block, one an hour after and the second one 15 minutes after, the explanation of which deaths I will give later on; and finally, 2 had their previous pathological state aggravated. If we subtract these 14 cases from the total of 34, there are left 20 cases whose status was bettered by the

TABLE I.

Left hemiplegias	12
Right hemiplegias	8
Left hemiparesis	2
Right hemiparesis	10
Lacunar spasmodic sclerosis	1
Menopause troubles	1
Essential amnesia	1
Softening of the brain	1
Cerebro-sclerosis	1
	37

Arterial hypertension:	
20 out 37 cases	54.2%
Stroke	8
Without stroke	26

TABLE II.

Concomitant lesions:	
Aphasias	8
Dysarthrias	3
Amnesia	3
Agraphia	1
Agraphia, vaso spasm of the Sylvian artery	1

Intervals between the attacks. From 1 day to 5 years and the stellar blocks.

9 patients blocked from 1 day to 17 days after onset of disease

1 patient blocked after 5 weeks after onset of disease

16 patients blocked from 1½ months to 22 months after onset of disease

11 patients blocked from 1 year to 4 years after onset of disease

TABLE III.

IMPROVEMENT IN THE CONCOMITANT LESIONS

Aphasias	4 out of 8..	50%
Dysarthrias	2 out of 3..	66%
Amnesia	1 out of 3..	33.3%
Agraphia	1 out of 1..	100%
Vasospasm of the Sylvian artery	1 out of 1..	100%

Menopause troubles—Improvement	70%
Failures:	
Temporary partial regression	3
No improvement of *statu quo*	7
Death after block	2
1st one—15 min. after block	
2nd one—1 hour after block	
Aggravation of the pathological previous condition	2
	14

TABLE IV.

No answer from the questionnaire sent to the patients	3
General end-results	
100%	6
90%	2
80%	4
75%	2
70%	1
50%	2
30 to 40%	3
	20

TABLE V.

Cases treated	37
Cases blocked and followed up	34
Improved cases and cures	20
No improvement	14
Percentage of the positive end results	61.8%

stellar block, giving a percentage of about 61% of positive results. In all the 20 successful cases there has never been any return of the vaso-spasm, of the hemiplegia or of any vasculo-cerebral pathological manifestation up to the present time (see Tables I, II, III, IV and V).

I do not believe in stressing the high therapeutic worth of the stellar block in cerebral vaso-neuro-trophoses, the original conception of which belongs, for the major part, to Professor Léger, my most faithful and fervent associate. Now that we have acquired some experience regarding these infiltrations, we tend, at present, to select the patients who are to undergo the block. At 70 years or after, we warn the person and his relatives of certain dangers, such as heart collapse, bulbar inhibition or risky sympathetic reaction. As for myself, I do not block too recent hemiplegias, on account of the possible renewal of the hæmorrhage through acute vaso-dilation. I wait three or four weeks before undertaking the injections where the palsy seems definitively fixed. Fresh constituted softening of the brain can be treated by this method but without promising more than we give. The consolidated hemiplegias dating from 6 months to several years will benefit by this therapy because of the considerable amount of vaso-spasm which provokes continuous reflexes and one will have the same surprise, as I often have had it, at the amelioration of the symptoms and even their disappearance.

The concomitant aphasias and dysarthrias will be improved in a frequently astonishing proportion. I assume that the hemiplegia depends upon the pathological condition of the artery but, once established, it is always aggravated by the vaso-constriction, such as the pains which the patients feel in their head and in their affected limbs. After they have been blocked, these sufferings cease and it is one of the reasons why they recover the use of their arms and legs whilst the superior centres are freed and resume a part or all of their functions through re-canalization of the vessels, or through the arterial neo-collateralization provoked by the sympathetic block.

This statement is so true that most of the pathological vascular brain conditions could perhaps be improved through the action of nupercain only. There is an impression the effects of nupercain are of a very temporary nature, lasting only 24, 36 or 48 hours. This is not my experience. I remember a case of angina pectoris, with repeated attacks, which was completely stopped by a single stellar infiltration. This happened 7 years ago and the spells have never returned since. I could quote several other cases of lumbar block which went on the same way.

But I immediately see the objection that the nervous cells, once destroyed, do not regenerate and are placed in neuroglia. Can the provoked cerebral vaso-dilation determine the *restitutio ad integrum* in the demolished cell? Is it not rather that most of the cases are affected with cerebral sclerosis, complicated with a greater or lesser arterial occlusion, which turns loose reflexes due to vaso-constriction? Again is it not the hypertension that is concerned, thus proved by the 20 cases out of the 37 that I have in files? We face usually a combination of both hypertension and sclerosis with vaso-spasm grafted on them and this could explain why my test operated successfully in the mentioned cases which were those where the nervous cells are inhibited. The function only was disturbed in the diseased artery. When there is no stroke, the circulation can be restored fairly quickly, and sometimes immediately after the block.

Let us conclude from the above statement that vaso-spasm is the basis of all the pathological phenomena related to the dysfunction, the unsteadiness or the occlusion of the cerebral circulation.

CAUSES OF DEATH IN THE PROVOKED VASO-DILATION

The primary effect of nupercain is one of vaso-constriction and secondarily it brings on a vaso-dilation. On the other hand, some subjects might naturally be sensitized to its action or might present a general debilitation out of their cerebral pathological status which might enhance the reaction. When this reaction occurs, I have observed an abatement of alertness in the patient. He becomes motionless and speechless, cyanotic, with a cold perspiration all over the body whilst the hands and feet are frigid; the pulse is fast and thready and diaphragmatic respiration decreases, the heart's beats diminish, all symptoms ending fatally if the patient does not return to normal. These troubles are too sympathetic-like not to arise from that system.

I have long been puzzled as to the causes of death in these cases. I might offer the following explanations for the 2 fatalities and the 3 sympathetic reactions which affected some of my patients in my series of the stellate infiltration.

1. The inferior cardiac nerves give off anastomoses with the laryngeal recurrents and the pneumogastric and a great many of its branches join the phrenic nerves which control the diaphragmatic contractions. Some of their branches serve to form the peri-bronchial plexuses, impulses from which lead to vaso-constriction of the pulmonary vessels and then to pulmonary ischæmia.

2. We all know the importance of the coronary vessels' innervation, born out of the inferior cardiac nerves and their collateral rami.

3. We might also take into account the prevertebral sympathetic inter-connections which are the thoroughfares conveying impulses to the counter-lateral chain and ganglia.

4. The organo-vegetative centre of the brain-stem, both motor and sensory, is irrigated by arteries proceeding from the vertebrals and their subdivisions. Nupercain injections in the immediate vicinity of these vascular formations produce their vaso-constriction which leads, when too long continued, to an acute anæmia of the medulla, necessarily spreading to both organo-vegetative and cerebro-spinal cardio-respiratory centres, thence failure of the respiration to end in a gradual cessation of the cardiac pulsations.

It might be a combination of these four elements which determined the two fatalities or the hypothymias after the injections in several cases but I believe that the severe vaso-constriction of the vertebral arteries was the main cause of death and reactions.

TREATMENT OF ESSENTIAL MENTAL TROUBLE

Since the stellar infiltration provokes a corresponding cerebral vaso-dilation; that this nupercain or nupercain-alcohol vaso-dilation changes the circulatory status of a given region; and that it may also, in fact, alter the spinal or para-sympathetic flow of impulses to the brain. I think I am justified in proposing to the neurologists to submit patients affected with essential mental troubles to the provoked *cerebral vaso-dilation test.*

Some might object that if the number of vascular ramifications is limited, it is not clear how the imperfectly irrigated territory could gain through provoked vaso-dilation. I will answer that this sort of vaso-dilation, from the induced blood flow, and perhaps through the effected stasis, will "urge on" new collateral formation so to create an increased drainage route. Is it a misconception that an unknown factor makes its way antidromically or otherwise after an excitation born from the cord or from the endocrine glands to stimulate the creation of new collaterals? It is one amongst the thousand means that nature puts at work to satisfy, through a concealed mechanism, the compensation law, of which so many examples are found everywhere. Experimentation will, perhaps some day, confirm this personal theory. Hypothesis today, truth to-morrow. The mind does not reject such a conception in persuance of the unswerving harmony that rules the various, supple and adaptable occult forces of nature.

If, for one moment, my proposition is accepted, the part of the neo-collateralization becomes evident. The more copious blood irrigation will fertilize the regions functionally sterile so far, will arouse the intellectual mission with the cellular life necessary materials supply, will rectify the nervous influx disturbances, will canalize the electrical potential which will convey more regular waves, will restore the customary balance of the higher centres.

What a vast and a promising field for neuro-surgical experimentation. An almost completely harmless treatment this stellate ganglion infiltration, a treatment full of suggestion and alluring possibilities! What will be its results? In this unexplored sphere the results, I venture to hope, will respond to my expectation.

May I now beg for the neuro-psychiatrists' co-operation? I hope to show in the future, favourable changes in a class of patients doomed, until the present time, to mental degeneration and to insane asylum commitment.

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A SIMPLIFIED PLAN OF PRESCRIBING FOR ADULT DIABETES*

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PRESCRIBING for a diabetic is interesting if the plan is simple and convenient. Otherwise it is a forced labour. The method we have evolved simplifies dietary prescription and offers a methodical directive to dispensing insulin dosage.

FUNDAMENTALS

The rationale of the treatment adheres to accepted principles. Derivatives realized from established rules govern the plan. The following remarks are reminders: the figures are explanatory, they do not burden later calculations.

For *normal,* healthy people the relative proportions of the chief nutrients are commonly quoted as: carbohydrates 4 gm., proteins 1 gm., fat 1 gm. (4:1:1). Five times the amount of total available carbohydrate is present to counterpoise the available fatty acids (5G:1FA).

For *minimal* limits securing sugar-fat conjugation the chief dietary constituents align themselves in these proportions: carbohydrates ½ gm., proteins ½ gm., fat 1¼ gm. The formula yields 2/3 parts of total available carbohydrate to 1 part of available fatty acids, which equals 1G:1.5F.A::(2/3G:1FA).

For *diabetic* use a wholesome formula which satisfies the usual obligations is: carbohydrates 1½ gm., proteins ¾ gm., fats ¾ gm., antiketogenic value (3G:1FA).

In a diabetic diet it is obligatory to have enough carbohydrates to protect against ketogenesis, enough proteins to promote growth and replacement, and a matched quantity of fat for fuel to supply the needed energy. A surplus of carbohydrates provokes pancreatic insular activity. A slightly greater amount of proteins than the minimal of ½ gm. per pound of body weight satisfies the meat habits of our people. Excess fat deposition in the liver especially, must be avoided. These allowances make possible an easier planning of a well balanced, attractive menu. The desirable objective is a menu approximating that which the average normal person uses. If the aim is to approach close to the normal, to concoct a sick man's diet is out of place. There is a difference between that which a healthy person can consume and what he gets accustomed to by habit, preference, domestic and clannish customs. Some sections of the population indulge in starches, others in meat and many northerners in fats. Compared with a high normal sugar consumption (5G:1FA) the diabetic formula (3G:1FA) is not far below average usage and it is still greatly in excess of the lowest compatible demands (2/3G:1FA).

DIETARY PRESCRIPTION

The first step in outlining a diet is to decide the amount of calories with which to provide the patient. The second step is to provide balanced portions of carbohydrates, protein and fats. Both steps merge into one in our plan.

It may be desired to begin with strictly basal requirements. That is only necessary when the patient cannot eat, or when forbidden to eat for other non-diabetic reasons, or when a strict weight reduction regimen is inaugurated. Otherwise no diabetic person in any stage who has an appetite needs to be launched on a starvation menu to begin the regulation ordeal. A moderate grade diabetes will not get worse on balanced rations of liberal food allowances. The severe diabetic must have insulin and that can be made to care for generous but balanced food allowances. Therefore it is reasonable to make a pleasant beginning, seldom using minimum basal diets. It is only useful to calculate basal requirements for the ideal weight, age and sex for another reason. The information serves as a base-line by which comparisons are made indicating how close or how far future amounts stray. For this estimation the Aub-DuBois scale of body surface area basal caloric gradations are standard. This computation can be made at leisure and one does not have to carry the graph and figures around.

At least approximate estimates of the expected weight should be made. Better than guess-work is our device: 100 lb. plus 3 lb. for each inch over 5 ft., plus 1 lb. for each year over 15. This is only more faulty, compared with the notorious difference of standard tables, at both extremes of height and age.

In youth caloric requirements per square metre of body surface are highest. In the adult the spread is not tremendous. Increasing years from 20 to 60 accompanied by the natural mounting weight is equalized by a lowered metabolism and there is little difference in the total calories.

20 yrs. at 5 ft. = M 1,475 Cal.: F 1,370 Cal.

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60 yrs. at 5 ft. = M 1,445 Cal.: F 1,350 Cal.
Increased height with proportionate weight for size in the same age bracket commands a wider spread.
20 yrs. at 6 ft. = M 1,870 Cal.: F 1,730 Cal.
60 yrs. at 6 ft. = M 1,890 Cal.: F 1,680 Cal.
The figures illustrate the basal number of calories for 24 hours at these age limits and at the average statistical weights, computed on the basis of body surface area for the two sexes.

Examination of the totals cited makes it apparent and plausible why we automatically drifted to a uniform beginning in all patients commencing correction of their unbalanced diabetes. We start all adults, with few exceptions, on an 1,800 calorie prescription (see Table I).

TABLE I.

BASIC DIET—1,800 CALORIES

		C. *175*	*P.* *85*	*F.* *85*
BREAKFAST				
Fruit	1 portion or equivalent	10.0	1.0	
Cereals	1 portion or equivalent	10.0	2.0	
Eggs	2 grade A or equivalent		12.0	12.0
Bacon	as allowed later			
Bread	1 slice (30 gm.)	18.0	3.0	
Dairies	1 pat (10 gm.) butter			8.5
	6 oz. (1 glass)	9 0	6.0	7.5
	½ oz. 19% (2 tbsp.) cream or equivalent	0.5	0.5	3.0
Coffee	as desired, no sugar			
NOON				
Soup	Fat free broth			
Meats	2 oz. 2 portions or equivalent		14.0	6.0
Vegetables	2 portions or equivalent	10.0	2.0	
Bread	2 slices bread or equivalent	36.0	6.0	
Dairies	1 pat butter or equivalent			8.5
	½ oz. 19% cream (1 tbsp.) cream or equivalent	0.5	0.5	3.0
	6 oz. (1 glass) milk or equivalent	9.0	6.0	7.5
Dessert	1 portion fruit or substitute	10.0	1.0	
Beverages	Tea, coffee—no sugar			
SUPPER				
Soup	Fat free broth			
Meats	2 portions or equivalent		14.0	6.0
Vegetables	2 portions or equivalent	10.0	2.0	
Bread	1 slice or equivalent	18.0	3.0	
Dairies	1 pat butter or equivalent			8.5
	½ oz. cream or equivalent	0.5	0.5	3.0
	½ portion 3 oz. milk or equivalent	5.0	3.0	4.0
	½ portion cheese	0.5	3.0	
Dessert	1 portion fruit or substitute	10.0	1.0	
Beverages	Tea, coffee—no sugar			
LUNCH				
Bread	1 slice or substitute	18.0	3.0	
Butter	1 pat			8.5

It may happen occasionally the amount is greater or smaller than it should be at the start; that is offset by the same chance the underweight and the overweight persons respectively will begin in the direction of the ultimate intention.

This is a workable medium amount. No diabetic patient has gone into coma because of this volume in correct proportions. If he goes into coma it is because he was already on the verge of coma. In that event even complete starvation will not likely prevent coma developing, but insulin will. In a day or two it is learned whether the amount is sufficient or insufficient. The increase or decrease as may be required is ordered beginning with 10% of the amount (1,800 calories), above or below the initial order. Further changes by the same step of addition or subtraction (see Table II) are made on sub-

TABLE II.

ADDITIONS (OR DEDUCTIONS)

		C.	*P.*	*F.*
1,980 CALORIES		*200*	*90*	*90*
10% Addition + (180 cal.)				
Bread	½ portion or equivalent	9.0	1.5	
Cereal	1 portion or equivalent	10.0	2.0	
Fruit	½ portion or equivalent	5.0	0.5	
Dairies	½ pat butter or equivalent			4.0
2,160 CALORIES		*C.* *230*	*P.* *95*	*F.* *95*
20% addition + (360 cal.)				
Cereal	1 portion or equivalent	10.0	2.0	
Fruit	1 portion or equivalent	10.0	1.0	
Bacon	1 portion bacon or equivalent		2.0	7.0
Bread	2 slices or equivalent	36.0	6.0	
Dairies	½ oz. cream or equivalent	0.5	0.5	3.0
2,340 CALORIES		*C.* *250*	*P.* *100*	*F.* *100*
30% ADDITION + (540 cal.)				
Fruit	1 portion or equivalent	10.0	1.0	
Vegetables	½ portion or equivalent	2.5	0.5	
Bread	3 slices or equivalent	54.0	9.0	
Dairies	½ pat butter or equivalent			4.0
	½ oz. cream or equivalent	0.5	0.5	3.0
	6 oz. milk or equivalent	9.0	6.0	7.5
2,520 CALORIES		*C.* *305*	*P.* *100*	*F.* *100*
40% ADDITION + (720 cal.)				
Cereal	1 portion or equivalent	10.0	2.0	
Fruit	2 portions or equivalent	20.0	2.0	
Vegetables	2 potatoes 2½ oz. each or equivalent	30.0	3.0	
Bread	2 slices or equivalent	36.0	6.0	
	3 crackers or equivalent	13.5	1.5	
	4 teaspoonsful jam	20.0		
Dairies	1 portion cream 19% (2 tbsp.)	1.0	1.0	6.0
	1 pat butter			8.5

TABLE III.

PORTION EQUIVALENTS

Cereal equivalents—Food value: carbohydrates 10; protein 2; fats 0.

Sample portion oatmeal cooked 1/3 cup.

Dry oatmeal, healthmeal, etc., 2 tbsp. (15 gm.); cooked oatmeal, healthmeal, etc., 1/3 cup (90 gm.); dry farina, cream of wheat 1½ tbsp. (13 gm.); cooked farina, cream of wheat 1/3 cup (90 gm.); shredded wheat ½ biscuit (13 gm.); all bran ¼ cup (16 gm.); bran flakes ½ cup (14 gm.); rice crispies 1/3 cup (11 gm.); corn flakes ½ cup (12 gm.); puffed rice ¾ cup (12 gm.); puffed wheat ¾ cup (13 gm.); pep ½ cup (13 gm.); dry cornmeal 2 tbsp. (15 gm.); cooked cornmeal 1/3 cup (90 gm.); ½ bread portion or equivalents, one portion of fruit or equivalent.

Bread equivalents—Food value: carbohydrates 18; protein 3, fats 0.

Sample portion 1 slice of bread ½" off 1 lb. loaf.

One small potato, 3 1/3 oz.; 1 glass of ginger ale 6 oz.; 4 soda biscuits or 2 arrowroots or 2 graham wafers; 3 rye wafers (3½" x 1¾" x ⅛"); 4 level teaspoons of jam or marmalade; 4 level teaspoons of sugar or honey; 6" cob of corn or ½ cup kernel corn; noodles or spaghetti ½ cup raw or ¾ cup cooked; rice 1/3 cup raw or ⅞ cup cooked; 1½ level tbsp. minute tapioca; macaroni ¼ cup uncooked or ¾ cooked—slightly less than two portions of cereals or equivalents, slightly less than two portions of fruits or equivalents.

Fruit equivalents—Food value: carbohydrate 10; proteins 1; fats 0.

Sample portion 1 orange (3 oz. wt.)

Apricots, fresh 2 large (80 gm.); applesauce 1/3 cup (85 gm.); banana ½ small (45 gm.); blackberries 1 cup (120 gm.); cantaloupe ½ medium (4" diameter, 130 gm.); grapefruit ½ size 80 (120 gm.); grapefruit juice ½ cup (100 gm.); honeydew melon 1 wedge 3½" of 6" melon (250 gm.); orange 1 whole size 220 (90 gm.); orange juice ½ cup (100 gm.); peach 1 medium (100 gm.); pear 1 small (90 gm.); pineapple, small fresh ¾ cup diced (100 gm.); pineapple juice 1/3 cup (80 gm.); black raspberries 2/3 cup (80 gm.); red raspberries ⅞ cup (100 gm.); strawberries 1 cup (100 gm.); watermelon large serving (150 gm.); tomato juice 1 cup (240 gm.); tangerine 1 large (100 gm.); rhubarb ½ cup (100 gm.); prunes 3 small prunes with 1 tbsp. juice (70 gm.); cherries ½ cup (100 gm.); grapes 10 average size (70 gm.); apple 1 small (85 gm.); ½ of bread portion equivalents. Fruit may be fresh, dried, or stewed and canned without sugar.

Vegetable equivalents—Food value: carbohydrates 5; proteins 1; fats 0.

150 gm. or 5 oz. of the following or approximately 1 cup:

Lettuce, cucumbers, spinach, asparagus, rhubarb, endive, marrow, sorrel, sauerkraut, beet greens, dandelions, swiss chard, celery, mushrooms, tomatoes, water cress, sea kale, cauliflower, egg plant, cabbage, radishes, leeks, string beans, young broccoli, French artichokes, green peppers, summer squash.

75 gm. or 2½ oz. of the following or approximately ½ cup.

String beans, brussel sprouts, pumpkin, turnips, squash, okra, beets, carrots, onions, green peas, very young.

Meat equivalents—Food value: carbohydrates 0; proteins 7; fats 3.

Sample portion 1 oz. lean meat.

Meat—lean cooked; chicken, all classes; fish, all classes; sardines, 30 gm. (1 large or 3 small) take off 1/3 pat butter; eggs, 1 egg less 1/3 pat butter; cheese or equivalent 1 oz. add 1/3 pat butter. Bacon 1/3 oz. or 1 strip, take off ½ pat butter and add ½ portion of cheese or equivalent.

TABLE III.—*Continued*

Dairy equivalents—

Butter—Food value: carbohydrates 0; proteins 0; fats 8.5.

Sample portion 1 pat 10 gm.

Butter 10 gm. (⅝" x ⅝" x ⅝"); lard 8½ gm. (½ tsp.); cooking fat (suet) 8½ gm. (2½ tsp.); salad oil or olive oil 8½ gm. (½ tbsp.); mayonnaise dressing 11½ gm. (¾ tbsp.); cod liver oil 8½ gm. (½ tbsp.); cream 19% 1½ oz. (3 tbsp.); oleomargarine 10 gm. (2/3 tbsp.).

Cheese—Food value: carbohydrates 1; proteins 6; fats 0. Sample portion 1 oz. cottage cheese.

Cheddar cheese 30 gm. (1 oz.); take off 1½ pat butter; American cheese 30 gm. (1 oz.) take off 1 pat butter; peanut butter 15 gm. (1 tbsp.) take off ½ glass milk.

Milk—Food value: carbohydrates 9; proteins 6; fats 7.5. Sample portion 6 oz. whole milk.

Buttermilk 6 oz. add 1 pat butter; skim milk 6 oz. add 1 pat butter; evaporated milk 3 oz.; cocoa 1 tsp. and 5¾ oz. milk; postum 2 tsp. and 5¾ oz. milk.

sequent days or at intervals to meet the patient's appetite and well being with an eye on the scale. In this procedure, there are liberties of movement. For instance, the increase may be dispensed in proportions similar to the ratio of the nutrients in the original formula. If it is otherwise desired, more carbohydrates in known quantities are permitted without increasing the fats and/or proteins. If there is a demand for more fat double the amount can be added without increasing the carbohydrates, and there is still no danger of a reverse in the ketogenic-antiketogenic ratio. Readjustments in diet are achieved by rationing portions of common values (see Table III). The question of adjusting additional calories for energy expansion as for sedentary workers and labourers is unanswered by time-worn routine stipulations as 20, 30 or 40% increases over basal estimates. We proceed by dictates in each individual, guided by the need for (*a*) satiation of appetite, (*b*) maintenance of well being, and (*c*) controlled by the direction of the patient's weight. It is not peculiar to diabetes that the malnourished should be fattened and the excess carriers reduced. It is healthier for the diabetic to weigh slightly less than the optimum.

INSULIN DOSAGE

The need for insulin medication is not as difficult to decide as is the amount to prescribe when it is needed. For habitual use, delayed acting protamine zinc insulin is foremost. Regular plain or unmodified insulin is resorted to as an adjunct in treatment. There are exceptions, in older people or when small amounts are

sufficient plain insulin will do. If carbohydrate tolerance is sufficient to superintend a balanced diet of adequate caloric content, no other treatment is necessary. An insufficient diet, sparing insulin, may bring the blood chemistry within normal but if it reduces body efficiency, it is a mistaken kindness. Insulin is indispensable when the sugar tolerance is inadequate to control the blood chemistry using a suitable diet.

There is no precision method in dispensing the correct dosage of insulin in the commencement of treatment in a beginner. Measures based on the quantity of sugar lost in 24 hours or taking account of the carbohydrates consumed and apportioning insulin accordingly are impractical. Generally recourse is to hit or miss measures. It is more orderly to pursue the following plan. We find a morning sugar value of 160 mgm. or more per 100 c.c. of blood after a twelve-hour fast is an almost certain indication for insulin use. This measure it should be noted is not comparable with the same figure of blood sugar content post-prandially and the peak in the standard glucose tolerance tests. At the commencement of treatment in conjunction with the basic 1,800 calorie diet and the blood sugar over the above level, insulin therapy is warranted. There is no risk in a dose of 10 units of delayed acting insulin before breakfast, especially when it can be foreseen the diet will be greater in amount than the initial basic 1,800 calories. If the following morning urinalyses show a reduction in the amount of glycosuria and ketonuria, the diet may be advanced 10% if it is advisable to satisfy a need without temporarily increasing the amount of insulin. The result of the subsequent morning urine tests will serve as indicators of the state of balance and of the advisability for further increases in diet or insulin or both.

A safe provisional allotment of insulin for higher blood sugar levels is an additional ¼ unit P.Z.I. for every mgm. above 160 of the fasting blood sugar up to a total of 40 units of the delayed acting insulin. A larger single dose of the same insulin is insecure. If more insulin will prove more satisfactory. Separate syringes are used; combination enhances delayed acting properties. In that case, the addition of plain insulin is approximated on the strength of the diurnal glycosuria present. Of post-prandial glycosuria determinations, it can be said they are only necessary when confronted with an obstinate situation. The aim is always one injection of insulin a day, at times two are compulsory and rarely more except during complications. When the morning specimen after discarding the overnight urine becomes sugar-free a final estimation of the fasting blood sugar is made as a control in respect to a misleading renal threshold and to an undisclosed hypoglycæmia.

In this procedure at the same time as the determinations progress, while insulin dosage is being adjusted, the tolerance for both quantity of food and sugar is established, the hazards of impending acidosis or coma and insulin reactions are practically eliminated. Rapid clearance measures, by heroic insulin dosages and starvation diets court uncertainties. There is the constant threat of interchangeable hypoglycæmia and hyperglycæmia due to eager over-treatment for both conditions. Quick results are not enduring. Hyperglycæmia can be reduced and glycosuria caused to disappear almost at will, but the organism does not respond at the same speed. Readjustment of the rate of metabolism in the untreated case requires more time.

Two drastic interruptions in the routine of the diabetic person occur frequently. The breakdowns occur in connection with surgical operations and fevers. Under these circumstances a systematic conversion plan can take the place of indirection. In the stablized patient undergoing a surgical operation, automatically the amount of insulin already used is divided into four equal shares, plain insulin is substituted and administered every six hours. As long as food is withheld, supplying fluids parenterally is a customary procedure to combat dehydration. The amount of fluid administered varies. To the injected fluid may be added 5% glucose supplying 50, 100 or 150 or more grams of sugar in twenty-four hours. If the amount of glucose is not greater than the already known carbohydrate content of the diet, larger amounts of insulin may not be required. On the other hand, in acidosis and where there is a serious infection, larger amounts of insulin are called for to cope with the unpredictable refractory state. As soon as the patient is allowed to eat he may be served ¼, ½ or ¾ or the full amount of his former diet, or substitutes of parallel composition (fruit juices, gruels, etc.). At the same

time, the previous form of delayed acting insulin is resumed in single daily dosages reduced in corresponding amounts (¼, ½ or ¾ of original dose) to the percentage reduction in diet.

SUMMARY

The plan described demonstrates the feasibility of a uniform, balanced, liberal basic diet in the commencement of the regulation ordeal in the treatment of diabetes. The ingredients of the menu are computed on the basis of edible portions. The portions of equivalent values serve as units for replacements, additions or deductions. A minor discrepancy enters into the matter of mathematical precision in the calculations because the exact percentage composition of the various elements of nutrition are not wholly standardized. There is an uncritical margin of error in servings which rates up to 15% according to dietitians' estimates.

A guide in prescribing insulin is outlined. Also a conversion scheme is mentioned when it is necessary to withhold food from a diabetic person on account of a surgical operation or for other reasons.

In review 200 records of patients were examined. Of this number 129 began with the basic diet plan. Before this procedure was adopted 71 were started on the traditional basal calorie induction method. The net result was: 16 did not require insulin; 9 used two kinds of insulin; 143 used a single daily dose; and in 33 the treatment was delegated to others. It is of interest to have found the total caloric requirements fell short of the amounts customarily advocated as necessary for maintenance at work.

If a sulfonamide must be prescribed for a second time, a different compound should be used, especially if reactions appeared during the original sulfonamide treatment. After 78 patients had fever, dermatitis, or conjunctivitis during sulfonamide therapy, 33, or 69%, of 48 subsequently given the same drug were similarly affected. Harry F. Dowling, M.D., and associates of George Washington University, Washington, D.C., observed that only 5, or 17%, of 30 patients from the original group had harmful reactions to a different sulfonamide. Of 9 patients reacting adversely to both of two courses of treatment, 5 had toxic manifestations when the same drug was given a third time. Toxic response on retreatment fore no relation to the original number of reactions.—*Ann. Int. Med.*, **24**: 629, 1946.

CASE REPORTS

MUCINOUS ADENOCARCINOMA OF URACHUS, INVADING THE URINARY BLADDER*

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Like the urachal cyst, the mucinous carcinoma of the urachus is relatively rare, but it does occur, with extension to the urinary bladder, often enough for the urologist to be on the alert for its detection. Hayes and Segal, in 1945, reviewed 44 cases from the literature and added a case of their own (*Journal of Urology*, **53**: 5, 1945).

The anlage of the urachus is the primitive hind gut of the early fetus. The type of development of the cells is decided by the proximity to the site of the urorectal septum formation and that the cells retain totipotent germinal powers with a tendency to develop along their respective three primitive layers. In one-third of the anatomical dissections the urachus communicated with the bladder cavity; however, a transverse fold of the mucosa acts as a valve and only with great increase of intravesical pressure will urine escape past this barrier. The lining of the urachus usually consists of one to three layers of epithelium without a basement membrane.

In their review of the reported cases, Hayes and Segal found that, as with urachal cysts, males predominate in the proportion of about two to one. The majority of these tumours occurred in males in the middle and latter age groups. The youngest case on record was a twenty-six year old male. Of all the cases reported, only one did not show the neoplasm completely eroded through the bladder mucosa. On cystoscopy, these cases showed either a fungating ulceration or a papillary mass of varying size at the vault of the bladder.

J.S., a 73-year old male, was first seen in the Urologic Clinic at Shaughnessy Hospital on May 5, 1944, complaining of frequency days 6 to 10, nights 3 to 4 for the past 1½ to 2 years, much worse for the past week, and pain in the left loin and groin of several days' duration. He had passed no sand or gravel in his urine. There had been no weight loss that he had noted. His general health was otherwise good. His bowels were regular and his appetite good. Physical examination

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was essentially negative. Kidneys not palpable, but slight left costovertebral angle tenderness was present. The abdominal examination was negative. External genitalia were normal on palpation. Rectal examination showed good sphincter tone; hæmorrhoids present; no abnormality in the rectum noted on palpation. Prostate enlarged grade 1 plus, glandular, smooth, not tender, and not fixed. Residual urine 3 ounces. Excretory urogram showed apparently normal appearance and function of upper urinary tract. Marked osteoarthritis present. Cystogram was unfortunately cut off on all the films. Admission for prostatectomy was advised. Shortly after admission the patient developed an acute left epididymitis. Urine, dark amber, nsq., acid/leucocytes 4 plus, red blood cells rare to occasional albumen 3+, non-protein nitrogen 36 mgm. %, hæmoglobin 85%.

On June 1, a suprapubic cystostomy for drainage was done. A tumour the size of a golf ball was found in the wall of the vault of the bladder protruding into the bladder. This was widely excised. The prostate was noted as being moderately enlarged. A bilateral vasectomy was done.

Pathological report.—"A small sac-like structure the size of an almond nut from which protrudes a large papillary growth, 4 x 3 cm., which on section shows colloid appearance with fair circumscription of basal portion. The microscopic examination shows many tortuous, enlarged, aborted, frequently inter-communicating acinar formations lined by one or more layers of atypical columnar epithelium and in some areas there is a marked degree of mucoid or so-called 'colloid' change producing a typical picture of a colloid carcinoma. This does not suggest an origin from the bladder and more likely is either an extension or a metastasis of a colloid carcinoma of the gastro-intestinal tract. Sections throug the wall of the sac show loss of lining epithelium and diffuse lymphocytic and plasma cell infiltration throughout the wall and there is no suggestion of any malignant process. Pathological diagnosis is metastasis or extension of colloid carcinoma of the gastro-intestinal tract, probably rectum, colon, or cæcum to the urinary bladder."

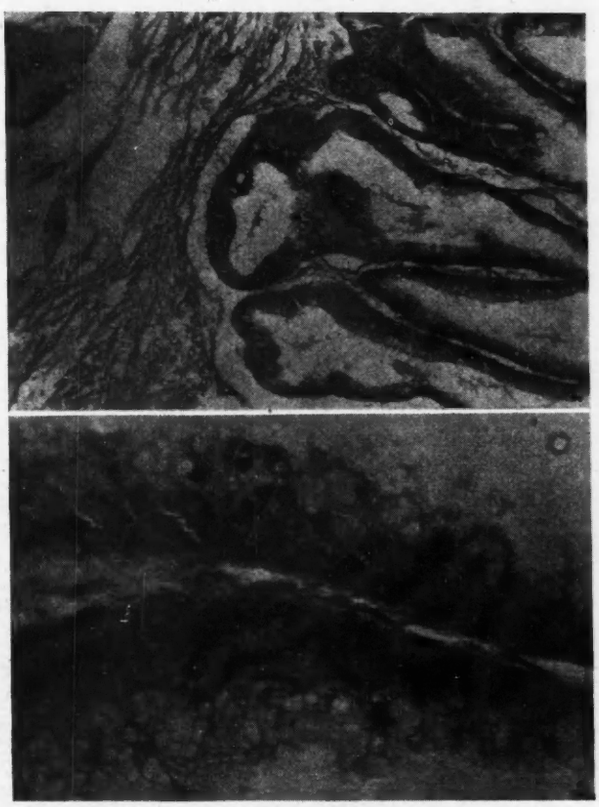

Fig. 1.—Magnification 10 x 6.
Fig. 2.—Magnification 45 x 6.

A barium series and barium enema were done but showed no apparent abnormality of the gastro-intestinal tract. Proctoscopic examination revealed no abnormality of the rectum or sigmoid.

Consultation report of the surgical specialist.—"The tumour is secondary to growth of the cæcum most probably, but a laparotomy is not advised due to the age of the patient and the presence of metastasis."

Accordingly, he was discharged from hospital. He was advised to return in four months for re-examination.

However, this patient did not return for follow-up examination until January 28, 1946, when I first saw him. He was complaining of frequency days 7 to 10, nights 3 to 4 of four years' duration, also marked difficulty in starting the urinary stream, and slowness in emptying his bladder, with a weak, dribbling stream. All these symptoms had become progressively more severe since his discharge from hospital in 1944. He had lost no weight and had not passed blood either in his urine or in his stool. His bowel habits were regular. His physical examination on admission was essentially negative except for a blood pressure of 180/90. No costovertebral angle tenderness and his kidneys were not palpable. The suprapubic incision was well healed and there was no evidence of local recurrence of tumour in the scar. No mass could be palpated on bimanual examination. The prostrate was enlarged grade 1 plus, glandular, smooth, not tender and mobile. Rectal sphincter tone was normal, hæmorrhoids were present but nothing abnormal could be palpated in the rectum.

The urine was amber, cloudy; 1.027; acid; occasional red cells; 3 plus white cells; albumen 3+. Culture showed *B. coli.* Excretory urogram showed apparently normal appearance and function of the upper urinary tract. Cystogram showed gross irregularity of the bladder contour and the floor of the bladder was elevated ½ inch above the symphysis pubis and a filling defect at the base was suggestive of shadow of prostatic intrusion. Marked osteoarthritis of lower dorsal and lumbar spine. No evidence of any metastatic lesion. X-ray of chest was negative. The cardio-thoracic ratio was 14.7 to 31 cm. Barium series and barium enema were normal and a proctoscopic examination revealed no abnormality of rectum or sigmoid. Cystoscopic examination of February 5, showed marked bladder trabeculation due to median bar type prostatic enlargement with grade 2 or 3 intra-urethral lateral lobe intrusion. Ureters were visualized but not catheterized. No evidence of tumour seen, although the site of the scar of the previous operation could be seen at the apex of the bladder.

I questioned the surgeon who performed the original suprapubic operation about the site of the tumour and sent a summary of the clinical findings to the pathologist who then kindly reviewed the microscopic slides of the growth with me. The pathological diagnosis was then changed to "Mucinous carcinoma of the urachus, invading the urinary bladder".

A per urethral prostatectomy was performed, and 18 gm. of tissue removed. Pathology report: "Benign prostatic hypertrophy".

The patient was discharged from hospital with a good postoperative functional result. He was instructed to report for cystoscopic examination in three months. He has been re-checked twice since then, the last examination being on November 12, 1946. There was no evidence of bladder tumour.

It is now almost three years since the bladder tumour was removed. There has been no sign of a recurrence of tumour. A review of the microscopic sections on January 2, 1947, leaves the pathological as well as the clinical diagnosis

unchanged. He is a three-year cure. It has been impressed upon the patient that he must return for re-examination every four months for the next two years and then once or twice a year.

This patient's only presenting symptoms were those of prostatism. The initial pain in the left loin and groin and burning on voiding were undoubtedly due to a prostatitis, vasitis and culminating in an acute epididymitis. It was probably for this reason that he was not cystoscoped prior to his operation. Undoubtedly cystoscopy would have revealed the tumour at the apex of the bladder. The patient at no time noticed blood in his urine. It was indeed unfortunate that the cystogram was cut off the films in the excretory urogram as they probably would have revealed a filling defect in the bladder vault that would have made the examiner suspicious of a tumour of the bladder before operation.

925 West Georgia.

A CASE OF SCHISTOSOMIASIS IN MANITOBA

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Until recent years so-called tropical diseases have received far too little consideration in Canada. A rude and costly awakening occurred when the South West Pacific campaign bogged down at first for lack of knowledge of malariology and other parasitic diseases. A belated correction of the deficiency of training in recognition and treatment of tropical diseases was then made in most of our medical colleges. Such special knowledge is still necessary because many ex-service men who served in endemic areas of these diseases will present themselves with late results of such infection. Enquiry into the geographical travels and sojourns of such patients will often indicate the true diagnosis.

However, this fortunate solution of the case will only be so if the medical profession here are tropically conscious. The present cases being encountered of recurrent malaria will taper off and cease.[1] Amœbiasis has received considerable notice recently and its late results will continue to be important.[2, 3, 4, 5] In addition the possibility of such diseases as schistosomiasis, clonorchiasis, filariasis, giardiasis, etc., and the residual effects of other tropical diseases must be remembered and understood.

The following is a case in point:

J.L., a white male, aged 68, a veteran of the Boer War, was admitted to hospital complaining of hæmaturia; burning sensation on micturition; suprapubic pain; pain in the right loin; frequency by day 1½ hours, by night 2 hours; loss of weight 15 lb. over a period of years.

History.—In 1903 in Northern Transvaal during the Boer war he developed hæmaturia and dysuria and was told by the M.O. that he had "bilharzia" (schistosomiasis). He gave an indefinite history of intermittent hæmaturia every fortnight or so ever since but did not consider it important enough to consult a physician. Recently increasing suprapubic and right loin pain led him to consult a physician and he was then admitted to hospital for investigation April 6, 1946.

Physical findings.—Examination showed a thin old man not obviously anæmic. No skin lesions. Head and neck negative. Thorax, no evident disease. Electrocardiograph "probably normal". Pain in right loin; pain and tenderness over pubes. No palpable masses.

Urinalysis during hospitalization showed gross blood and pus. Never any casts. Blood urea nitrogen on admission 15.8 mgm. %; before death 91.6 mgm. %.

Cystoscopy after admission revealed a "dirty bladder" with much phosphatic encrustation near the right ureteral orifice, which orifice could not be found. Subsequent repeated cystoscopy still showed no trace of the right ureteral orifice and intravenous pyelogram showed no sign of right kidney or dye excretion. The left kidney however was well visualized. Left retrograde urogram normal. Cystogram showed a double shadow resembling a diverticulum.

Digital rectal examination disclosed a flat firm mass above the prostate. Accordingly sigmoidoscopic examination was done and the condition was considered to be seminal vesiculitis. Further examination to rule out tumour was done. Barium enema was reported on as follows: Colon is negative. Cæcum not tender. Appendix is visualized. Barium series was done, with negative results.

Cystoscopy was then repeated before considering a right nephrectomy. Some of the encrusted material was scraped away from the region of the right ureteral orifice. Tissue from this area was sent to the pathologist and was reported as "papillary carcinoma". It was considered that further operation was inadvisable.

Patient steadily became more cachectic with frequency by day and night with some dysuria and gross hæmaturia at times. Transplantation of ureters to the exterior was considered inadvisable. After being semicomatose for a week, he died October 12, 1946.

Autopsy findings, gross.—Externally, emaciation, cachexia, senility beyond age. The pertinent findings were as follows: Consolidation of lower lobes of both lungs with commencing abscess formation. Heart showed minimal arteriosclerotic changes in the coronaries.

The abdominal viscera were fixed by recent adhesions and there was thick yellow-green pus in the cavity which appeared to come from the pelvic retroperitoneal area. The liver showed metastatic white tumour nodules 2 cm. in diameter. Kidneys were both hydronephrotic, with ureters dilated irregularly in places to 3 to 4 cm. in diameter.

The bladder was filled with cloudy urine and firmly adherent on all surfaces. The lining was dark, roughened, thickened and encrusted and much of it was involved by diffuse infiltrating tumour growth. This tumour growth invaded surrounding structures and penetrates deeply into the rectal wall.

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Stomach and intestines showed no evidence of disease except in the rectum as above noted.

Microscopic findings of note.—Heart: myocardial fibrosis. Lungs: bilateral lobar pneumonia with abscess formation. Occasional small scattered metastatic carcinoma in peripheral areas. Liver: metastatic carcinoma—papillary. Left kidney: section shows some degeneration and fibrosis of tubules. There are a few greatly enlarged amyloid glomeruli but most are normal. Arteries show arteriosclerosis. Right kidney: section shows marked atrophy of tubules with replacement fibrosis. A few tubules remain. All existing glomeruli are

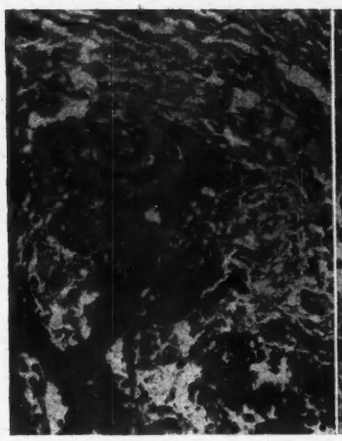

Fig. 1

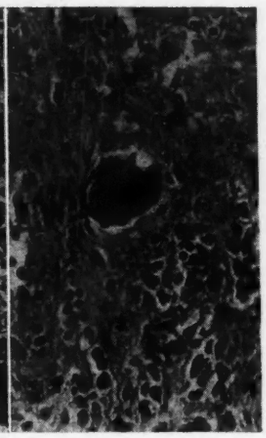

Fig. 2

surrounded by increased capsular space so that they appear to be set in cystic spaces. Other glomeruli have been replaced by fibrosis or hyalinized. There is minimal scattered cortical round-celled infiltration. Right ureter: sections show thinning and fibrosis of the wall with loss and hyalization of muscle elements and deficient epithelial lining.

Bladder: anaplastic papillary carcinoma widely penetrating the bladder wall (Fig. 1). Sections taken from the region of the vesical plexus show solitary schistosoma (bilharzia) ova here and there in the submucosa in a fairly good state of preservation and possessing a distinct postero-lateral spine as seen in *Schistosoma mansoni* (Fig. 2). The mucosa is lacking and the submucosa shows chronic inflammatory infiltration and fibrosis.

DISCUSSION

This case well illustrates that "what one knows one sees". The history of bilharzia in this patient was dismissed by the clinician in his notes as of no consequence. Yet if any of the clinicians consulted by a man with so long a history of hæmaturia had properly treated the schistosomiasis (bilharziasis) he would not have developed schistosoma cancer of the bladder, from which the eventual termination of peritonitis was to be expected. That carcinoma is to be expected in schistosomiasis is well known, and the incidence increases with age as would be expected.[8, 9] Treatment of schistosomiasis (bilharziasis) with very satisfactory results has been reported on from time to time.[6, 7]

The microscopic appearance of the egg as seen in Fig. 2 clearly indicates *Schistosoma Mansoni* which is endemic in the Transvaal but usually infects the intestinal rather than bladder walls. That it did not do so is evident from the negative sigmoidoscopic and x-ray findings and absence at autopsy of intestinal lesions. It is interesting that the ova, of which several were seen in sections, were not calcified, indicating that the parasite probably deposited them in the not distant past and that these blood flukes may live long in the veins of man.

The infection occurs in endemic areas which are now fairly well demarcated, chiefly in Africa and adjacent Madagascar, Arabia and Palestine, in tropical South America and in Japan, China, and islands of the southwest Pacific. The infecting agent is a short-lived swimming cercaria which is found in quiet waters harbouring the secondary snail host, and penetrates the bare skin of those entering such waters or the buccal mucosa if water is drunk. It travels by the blood stream to the heart, then on through the lungs and finally by the portal system to the liver in which location only can maturity be developed. Thence the male and female paired worms travel against the current up the venous bifurcations to arrive either in the intestinal venous plexuses or the vesical plexus.

It seems that earlier production of viable ova by *Schistosoma japonicum* induces the female to locate after a shorter journey from the liver, hence they are found almost always in the intestines and not infrequently in the appendix. *Schistosoma mansoni* matures a little later and though usually in the intestines may reach the vesical plexus and the ova appear in the urine. *Schistosoma hæmatobium* maturing the latest almost invariably is found in the vesical walls. Ova are laid in minute veins distended by the gravid female and these spined eggs by help of histiolytic action gradually work their way through the specific intestinal or vesical walls aided by muscular action to eventually penetrate the submucosa and mucosa and reach an avenue of escape in fæces or urine. Only when diluted by water will the ova hatch and in this water the motile embryo swims to find the appropriate snail in whose body it develops and multiplies a thousand-fold, later to emerge as fork-tailed swimming infective cercaria capable of penetrating the unbroken skin of man or animals.

Avian schistosomes exist in North America which are not infective to man but whose cercaria do penetrate the skin of bathers and

are a potent source in the United States and Canada of "swimmers' itch".

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ACUTE APPENDICITIS WITH GANGRENE OF THE SIGMOID COLON

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Having been unable to find in the literature any instance of sigmoid gangrene due to acute appendicitis, it is felt that the following case would be of surgical interest, and the method of treatment, which happily led to a satisfactory result, would be of benefit. The major decision as to the plan of procedure took place during the operation, at which time the possible degree of mortality was carefully considered.

On April 17, 1946, a telephone call was received from the country, saying that a boy of fourteen was coming in immediately, with severe pain in the right iliac fossa. Office examination revealed a history of an attack of severe lower right abdominal pain, which was now in its fifth day. A dose of epsom salts had been given at the outset, but as the bowels did not move and the pain did not decrease, a heavier dose of salts was given the following day. This was followed by vomiting, with no improvement. Salts again were given on the day following (Monday) when the bowels moved, with slight decrease in pain.

When I first saw him Wednesday afternoon abdominal examination showed marked right lower quadrant rigidity and tenderness, temperature 100, white blood count 19,000. He was immediately taken to the hospital and under general anæsthesia operation was commenced. Due to the half-day holiday, it was impossible to procure an assistant on short notice, and the operation was begun with the assistance of a nurse.

The appendix and cæcum were markedly œdematous at the base and the appendix itself could not be raised. The base of the appendix was tied and cut across, cauterizing with carbolic and alcohol. Working backwards, the mesentery was clipped and tied, the wound enlarged markedly toward the median line and right rectus muscle retracted medially. Finally, after blunt finger dissection, the end of the appendix, consisting of a mass 6 cm. in diameter, was elevated. It was then found that the gangrenous mass was intimately adherent to a loop of sigmoid colon, which also was gangrenous and completely obstructed. The area of gangrene in the sigmoid colon was approximately 2½ inches in length. The appendix was peeled off the sigmoid loop and removed.

The problem then was as to procedure, because no lumen could possibly be demonstrated in the sigmoid colon. It was felt that resection in the presence of such inflammation would be fatal. It was decided to exteriorize the gangrenous loop, after incising the left lateral border of the mesentery. The whole gangrenous mass was then brought out on the abdominal wall and the incision closed with steel wire, after placing penicillin powder and sulfathiazole in the pelvis. A Penrose drain was carried down into the pelvis. The following day the proximal part of the gangrenous loop was opened and a rectal tube inserted into the proximal part of the colon.

Despite sulfonamides and penicillin, the temperature became elevated considerably and distension developed, which required a Miller-Abbott tube. Finally, a large flow of pus presented itself on the right lateral side of the exteriorized loop. This continued for approximately ten days, and was followed by free fæcal drainage from this sinus, evidently coming from the cæcum, as was later proved by x-ray. At the time of the fæcal flow from the fistula the intestinal contents stopped and no results were obtained by enemata from the proximal sigmoid loop. When this fæcal flow commenced the patient's general condition improved markedly, and he was allowed to go home 2½ weeks following the operation. By this time, the anterior and lateral walls of the sigmoid loop had sloughed off, and there remained two intestinal openings, one proximal and one distal approximately 5 cm. apart, with mucous membrane connecting them.

The flow from the fæcal fistula continued throughout the summer until August 31, when it suddenly ceased and flow from the proximal sigmoid opening commenced. He was brought into hospital again and given sulfasuxadine in moderately high dosage for five days, with low residue diet. Operation consisted of freeing the intestinal loop down to the peritoneum, being careful not to open the peritoneal cavity. It was then found possible to invaginate the mass and suture the two openings in a transverse direction, after having inserted a soft rubber tube about four inches in length at the site of the closure. One layer of Connel stitch and three layers of Lambert sutures were applied, the last being interrupted silk. The fascia was brought together over the closed bowel with steel wire, and sulfamul ointment was applied to the area under the fascia and to the fat area. Two rubber tissue drains were inserted, one under and one above the fascia. He was kept on intravenous therapy for five days, with penicillin, and at the end of this time the tissue drains were removed. There was no evidence of infection or distension.

He was allowed to go home in ten days, with the wound completely healed, and was able to have normal bowel movements, with the aid of fairly large doses of mineral oil. The rubber tube was evacuated three weeks after operation, and to date there has been no disability or weakness in the abdominal wound. There is no resulting constipation.

The above case is of interest for two reasons: (1) the rarity of an obstructive gangrenous lesion of the colon as a complication of appendicitis; (2) the successful termination of the case, due primarily to the process of exteriorization and the use of penicillin and sulfasuxadine.

PNEUMOCOCCAL PERITONITIS WITH PERFORATION OF DUODENUM

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The following unusual case of pneumococcal peritonitis with perforation of the duodenum is recorded, as it proved to be of great diagnostic and surgical interest.

The patient, a white male of 27 years of age, was admitted to the Queen Mary Veterans' Hospital at

17:15 hours, January 12, 1947. He gave a history of being in perfect health until about suppertime on January 11, when, after finishing a bowl of soup, he developed a severe pain over the lower part of his abdomen. This pain then spread over the whole of his abdomen and anterior chest wall. Almost immediately he vomited several times and on one occasion the vomitus was black. He had been constipated for the past three days except for one small movement shortly after the onset of his abdominal pain. The patient at this time denied any previous gastric trouble or any recent illness but later recalled vague dyspeptic symptoms during 1946. Barium series in April, 1946, had shown no lesion.

Physical examination on admission showed a pale, sick looking patient. Temperature 99.4°, pulse 116, respirations 22, blood pressure 140/100. Abdomen distended and rigid, tenderness present everywhere but particularly in the right lower quadrant. Rectal: no masses but tenderness present in all quadrants at the apex of the examining finger. Chest clear to percussion and auscultation. Leucocytes 20,200, urinalysis negative. X-ray—antero-posterior and lateral views of abdomen showed no free air, no fluid levels.

Operation 22:30 hours on January 12; spinal anæsthetic. The abdomen was opened through a right paramedian incision and considerable greenish yellow fluid was immediately encountered and aspirated. Then it became obvious that all the loops of small bowel were plastered to one another and to the anterior peritoneum and to the liver with a very extensive fibrinous exudate. In the search for the lesion, many of these adhesions were broken down and loculated pockets of fluid were found throughout the abdomen. The appendix was found in its normal position and seemed to be innocent. The small bowel was examined from the terminal ileum up to the duodeno-jejunal flexure and no primary lesion was found. Similarly, a search of the large bowel from cæcum to rectum proved negative. The intestines were next packed off and a further pocket of necrosis plastering the stomach and duodenum to the liver was broken down. It then became evident that there was an area of necrosis about 2 cm. in diameter on the anterior wall of the first part of the duodenum and in the centre of this area was a small leaking perforation. The whole necrotic area had the appearance and consistency of the eschar of a burn of the skin. A culture was taken of the peritoneal fluid and discharge from the duodenal perforation.

Because of the surrounding œdema and necrosis and because the great omentum was so short, closure of the leak was difficult. However, a tag of great omentum was brought up and placed over the necrotic area and was fixed there with three mattress sutures of 00 chromic catgut. The abdomen was again sucked out and the whole wound was closed in layers without drainage. During the operation, the patient was given 1,200 c.c. 5% glucose saline and 550 c.c. blood intravenously.

January 13.—Laboratory report of culture of peritoneal fluid and exudate from site of duodenal perforation showed moderate growth of pneumococcus type 13, penicillin sensitive; light growth of green streptococcus, penicillin sensitive; very light growth *E. coli Cascaraba*. Continuous gastric suction via a Levine tube was instituted, with penicillin 50,000 units q.3h. and supportive intravenous fluids.

The patient developed a troublesome cough and sputum and blood cultures were taken. Chest x-ray on January 14 showed homogeneous density in both lower lung fields. Pneumococcus type 13 and hæmophilus influenza were isolated from the sputum.

January 17.—Further history obtained today reveals that the patient had had a severe upper respiratory infection on January 5 and 6 associated with considerable coughing but no other signs. Today the patient continues to show steady improvement, the temperature only rising to 100° and the pulse running at 72. X-rays of the chest on the last three days show continued clearing of the basilar cloudiness particularly on the left side. The percussion note is still dull and breath sounds are absent over the whole right axillary area.

January 23.—Two days ago and again today, the right chest was aspirated and total of 175 c.c. of amber fluid was obtained which proved sterile on culture. Blood culture negative. Sutures removed—primary wound healing.

There was steady progress from now on. Gastric analysis on February 6 with histamine showed a normal range of acid. Barium series showed scarring of duodenal cap with pseudo-diverticulum formation but no ulcer crater was seen.

DISCUSSION

The main discussion concerning this case centres in the perforation of the duodenum and evokes certain questions in one's mind. Was this the perforation of an ordinary duodenal ulcer or was the perforation the result of a septic infarct of the duodenum? In this connection, the belated history of previous vague gastric trouble must be recalled, and the fact that the patient had had a negative barium series nine months previously. And, more important, the duodenal lesion at operation did not present the clear punched-out ulcer that is so common in ordinary perforations.

Considering the development of pneumococcal peritonitis on this basis, it is conceivable that swallowed sputum could enter the peritoneal cavity through a perforated ulcer, and thereby set up a purulent peritonitis.

On the other side of the scale, there is the latent history of an acute upper respiratory infection less than a week before the acute perforation. Secondly, at operation, the necrotic area on the first part of the duodenum because of its size and consistency gave the appearance of an infarct that had broken down in its centre. To correlate these findings one could postulate that associated with the respiratory infection there had been a pneumococcal bacteræmia that had produced an infarct of the duodenum, and subsequent perforation and peritonitis. To substantiate this claim further is the x-ray finding of homogeneous density of both lower lung fields about twelve hours after operation, and the later pleural effusion on the right side. From the bacteriological angle a sputum culture one day postoperative also grew pneumococci type 13. Two blood cultures on successive days were sterile. Granted this would have clinched the etiological diagnosis if they had been positive, they were not, and therefore it is probable that the optimum time was missed.

Summing up the discussion, the weight of evidence favours the second theory. Therefore, this is presented as a unique case and in a

limited search of the literature, no similar record was found.

SUMMARY

A case of pneumococcal peritonitis and perforation of the duodenum with recovery is presented.

The etiology is presumptively the consequence of a transient pneumococcal bacteræmia.

CLINICAL and LABORATORY NOTES

SIMPLIFIED CLINICAL PHOTOGRAPHY

A. L. Yates, F.R.C.S.(Edin.)

Calgary, Alta.

Many doctors who wish to make photographic records of the clinical conditions that they meet with are deterred from doing so by the need of complicated and expensive apparatus, the use of which is time-consuming. The following method in which one merely points the camera at the patient and presses the button, has much to recommend it. The camera is carried in one's bag.

In the following description, it is assumed that the camera is focussed for a distance of twenty feet or more which is the case in all the cheaper fixed-focus cameras. The more expensive cameras which have a limited degree of focus are set to take a picture at this distance. The camera should have an iris diaphragm.

The method is based upon the fact* that if a lens of forty inches focal length is fixed with adhesive plaster or by other means in contact with the lens of the camera, the camera if focussed for a distance of twenty feet or for infinity is now in exact focus for objects which are forty inches from the lens. A lens of forty inches focal length is purchased from a manufacturing optician (Imperial or the Consolidated Optical Co.) or elsewhere, and is known as + I.D. One puts this lens in front of that of the camera, fixes it approximately centrally with adhesive plaster, measures the distance of the lens from the object which must be that of the focal length of the second lens, and takes the photograph. In the preceding table an attempt is made to indicate the extra lenses which may be required in different types of work. The letters in column 1 indicate the following: S. surgery; M. medicine; G. gynæcology; P. pædiatrics; R. rhinology; D. dermatology; I. ophthalmology; O. orthopædics; T. dentistry.

TABLE I.

Type of work	*Extra lens*	*Distance from object at which camera is held in inches*	*Size of object in inches*	*Size of image on film*
1. S.M.G.	+ 0.375. D.	100	72	2.8
2. P.D.	+ 0.50. D.	80	60	3.0
3. S.M.P.R. D.O.I.T.	+ 1.00. D.	40	20	2.0
4. P.R.I.	+ 1.50. D.	27	14	2.0
5. C.I.T.	+ 2.00. D.	20	10	2.0
6. I.T.	+ 4.00. D.	10	5	2.0
7. R.I.T.	+ 8.00. D.	5	2.5	2.0
8. I.	+10.00. D.	4	2	2.0

* To find the size of the image on the film: multiply the size of the object to be photographed, *e.g.*, 20″, by the length of the camera from lens to film, *e.g.*, 4″, and divide by the focal length of the lens applied in front of that of the camera, *e.g.*, 40″

$$\frac{4 \times 20}{40} = 2.$$

The preceding table refers to camera of the Brownie type which is four inches long from lens to film. If a smaller camera is used and is three inches long the size of the image will be found by dividing the figure shown in the last column by three-fourths. If a larger camera is used and has a length of five inches the figures in the last column must be multiplied by five-fourths.

In high summer the daylight provides adequate illumination. In winter sufficient illumination can be obtained by screwing a photo-flood lamp into the socket of the examination lamp connected to the main.

When the lens is held very close to the object, *e.g.*, 4 to 12 inches, the object which is seen in the centre of the view-finder will not be in the centre of the film. To overcome this difficulty, the lens of the camera should momentarily be placed opposite to and about one inch away from the centre of the object to be photographed. A fragment of adhesive tape should then be placed on the skin opposite to the view-finder. When this mark is seen centrally in the view-finder the object will be central on the film.

The method above described is based on a well known law of optics and has been employed for years in photography. It does not however appear to have been used in clinical photography which it simplifies.

Fermentation that takes place in the making of sauerkraut has little if any effect on the product's excellent vitamin C content.

THE CANADIAN MEDICAL ASSOCIATION

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EDITORIAL

ACUTE ANTERIOR POLIOMYELITIS

WORK in research on poliomyelitis continues daily and is supported by large endowments. Some progress has been made in understanding its pathogenesis and epidemiology. In spite of this work and the publicity given to epidemics, physicians sometimes fail to diagnose the disease. There are several reasons for this situation. The physician may have graduated without ever having seen a patient with acute poliomyelitis. This is no fault of our medical schools, as epidemics do not occur every year. In the past too much attention has been given to the fact that so-called flaccid paralysis of muscles is pathognomonic of anterior poliomyelitis. It does occur, of course, but more often we see weak, painful and tender muscles. Various muscle groups are more commonly affected than others, *e.g.*, shoulder girdle, quadriceps and dorsiflexors of the foot. Any muscle or muscle group may be affected and all must be examined carefully. During the epidemic last year, some clinicians saw patients with the disease months after the original infection, no diagnosis having been made. The diagnosis had been considered in most instances by the physician and patient or relatives, but not arrived at by the physician due to his failure to examine all muscles.

In many of the cases another factor was responsible for mistakes, namely, the patient was one of the first to be seen in the epidemic or one found at the end of the epidemic, or the case appeared as an isolated one in a community where the disease was not epidemic. However, in each instance too little attention was given to malaise with fever, headache, painful or "stiff" muscles (particularly muscles of the back) and a complete routine examination of all muscles. For example, in a few instances neglect in examining the abdominal muscles led to errors. Another patient had much back pain, then symptoms of an acute abdominal condition which proved to be an acutely distended neurogenic bladder, secondary to acute anterior poliomyelitis.

It is more important to examine function in muscles than to rely on presence or absence of tendon reflexes in a given case. It should be remembered that we do not examine routinely all muscles for reflex action. For example, reflexes in the biceps, quadriceps and calf muscles might all be normal and the patient have paralysis in other muscles. Spinal fluid examination will usually settle the diagnosis.

No one expects an epidemic this year, but isolated cases of the disease will occur. We must be alert to such a possibility. There has been much controversy over proper methods of treatment in the past few years. However, certain fundamentals are generally accepted and seem worthy of emphasis. Tender, painful muscles need rest and comfort. Heat in some form does give comfort. When this stage is passed, methods used in physical medicine, especially re-education of muscles, are of the utmost importance.

UNIVERSAL MEDICAL SERVICE IN NEW ZEALAND

OF all the democratic countries New Zealand has gone furthest in trying to work out a scheme of social security which includes the provision of medical service for the entire population. That country has had five years' experience of such a scheme, and a review of what has been accomplished is now given us in Dr. Douglas Robb's pamphlet "Health Reform in New Zealand."*

The history of the development of the present scheme has followed a familiar pattern. The plan for public organization of medical services under the Social Security Bill was undertaken by the first Labour Government in November 1935. This Bill was passed in 1938. The money for the implementation of the Act was raised by a new tax of 1 shilling in the pound payable by everybody, and has been collected since April 1939. This Act had two main objects:

*Health Reform in New Zealand, Douglas Robb, M.D., Ch.M., F.R.C.S.(Eng.), F.R.A.C.S., Whitecombe and Tombs, Ltd., Auckland, N.Z.

the replacement of existing non-contributory civil pensions by a system of monetary benefits on a contributing basis; and, the provision of a system of a general practitioner service available to all, as well as maternity benefits; pharmaceutical benefits, including all ordinary prescriptions and appliances; and hospital benefits which relieved the patient of a portion of such charges. Other benefits were to be added later such as x-ray, laboratory, massage, neurosurgery, district nursing and domestic service.

Apparently the Government worked out its scheme without consultation with organized medicine in the form of the New Zealand Branch of the B.M.A., which had itself proposed a scheme for a medical service involving a contributory form of insurance. The Government measure was political in conception and its contents were not made known to the B.M.A. till its introduction into the House. Later, however, it appears that negotiations were carried out and certain alterations in the benefits were made.

The general practitioner service was slow in being established, but is now in operation. The method of remuneration has five variations; a salaried whole time service; capitation; fee-for-service; a refund system whereby the patient pays the fee and gets a refund on his receipt later on; a "token" payment system or modified refund. The fee-for-service seems to be the method most widely followed. Specialist services have not yet been provided under the scheme, but a rebate of 7s. 6d. is allowed off specialist accounts for each attendance. Dental benefits were only inaugurated in February of this year.

One or two comments may be made. (1) As regards the financial aspects. To all intents and purposes the government is faced with an unlimited liability. As Dr. Robb puts it: "The fund has to be a bottomless pit." There is no doubt that the costs of medical service are rising very sharply. This is especially so in regard to the pharmaceutical benefit: in 1946 the annual cost per head for medicines alone was almost as much as that for general medical services. When other benefits come into operation, specialist etc., the costs will rise still more. (2) There has been no provision for urgently needed hospital expansion. With the rise in building costs this already acute problem has been rendered more urgent. (3) The quality of service rendered under general practice has not been improved. In Dr. Robb's opinion the fee-for-service method of payment lays the emphasis on quantity rather than quality and the result is quick superficial work. It is true that the average general practitioner is being very well paid, but it is at the expense of the best type of practice.

Dr. Robb is in full agreement with the principle of public responsibility for all health services, but he feels it should be pursued outside of the political sphere. "The professional groups with their inside knowledge of what could be done, and the political section with its desire to serve the people and implement such plans should be united, and if so would be irresistible. The measure of the divergence of viewpoint between the two groups is the measure of our failure as an intelligent society."

It would be better he feels, if action was withheld for some years and replaced by education of the people as to what can be done. There is much to be said for this point of view, but what political party would wait for such education? One point not brought out as clearly by Dr. Robb as we think it should be, is that a good universal health service is a costly affair, whether it is undertaken by private enterprise or government direction. In political plans much emphasis is laid on provision of medical care for all; too seldom is it understood that the quality of that care, indeed its very existence, depends on training that is expensive, equipment and establishments that are still more expensive, and research which is most expensive of all. By careful planning waste can be eliminated and spreading of costs can be worked out but the cost will still be there.

New Zealand now appears to be committed to this policy of universal free [so-called] medical service, and there is no indication that any basic alteration will be made. The later course of events will be interesting. Will the medical services be made more freely available? Will the standards be kept high? Will the cost of the scheme when fully developed be kept within reasonable limits?

EDITORIAL COMMENTS

Parergon

There should be little need now to draw attention to this well-known collection of artistic work by medical men. The present edition—the third—is a sheer pleasure, as were the two preceding ones. That it is more than double in size is some indication of the keen and rapidly developing interest in art amongst medical men. No doubt there has always been a certain amount of artistic skill in the profession, but by the unwearying efforts of Messrs. Mead Johnson self-expression has been encouraged in a large and steadily growing number of physicians. The examples so strikingly arranged in this volume (and the arrangement is in itself a high artistic accomplishment) are only selections from the exhibitions which are now accepted as an essential item on the program of large medical conventions.

Some day perhaps it may be found possible to reproduce in colour but that is a high ambition. In the mean time the American Physicians' Art Association is preparing to take over entirely the administrative work which has for long been so generously assumed by Messrs. Mead Johnson. Under the presidency of Dr. Harvey Agnew whose artistic skill and versatility are so outstanding we look for a vigorous and sustained expansion of the work of the American Physicians' Art Association.

Two New Professors in the University of Toronto

Recent appointments in the Faculty of the University of Toronto involve some striking personalities in Canadian medicine. Dr. Duncan Graham retires from a long and distinguished career as Professor of Medicine. It should be added that his services in the interests of the Association deserve similar characterization both for their devotion and their outstanding quality. He is to be succeeded by Dr. Ray Farquharson who has risen in his profession with a steady progression of honours and responsibilities which are naturally to be associated with a fine personality and the energy of a keen and well trained mind.

On the surgical side, Dr. W. E. Gallie retires from the professorship of surgery, with an international reputation in surgery and the affection of many surgical pupils. He is to be succeeded by Dr. Robert Janes, who has already gained great distinction for his work in chest and orthopædic surgery.

The University of Toronto may well congratulate itself on its choice for such important posts.

Third American Congress on Obstetrics and Gynæcology

This congress will be held in St. Louis, Miss., from September 8 to 10, 1947. We are asked to announce that an invitation is extended to members of the Obstetrical and Gynæcological Society of Canada to attend this meeting. There are several Canadians on the program, and the subjects to be dealt with will interest not only obstetricians and gynæcologists but general practitioners, hospital administrators and nurses. The program will include sections for the public health doctor and nurse. Round table discussions, demonstrations, and scientific and moving picture exhibits will complete what is expected to be an outstanding occasion.

MEN and BOOKS

JEANNE MANCE

H. E. MacDermot, M.D., F.R.C.P.[C.]

Montreal, Que.

Perhaps no other period in early Canadian history is called "romantic" more often than is that during which the French settled themselves along the valley of the St. Lawrence. The romance largely depends on how closely we inspect events, but there always is fascination in the extraordinary spirit of the men and women of the time.

The figure of Mademoiselle Jeanne Mance is prominent amongst these. The story of her life has an attraction of its own, but she also occupies a special place in the history of Canadian nursing. In her solicitude for the sick she antedates Florence Nightingale by nearly two centuries. The work of the two women differed in many respects, but there was the common impelling motive. The one established under peculiar difficulties a hospital which has maintained unbroken her tradition of service to the sick poor: Jeanne Mance lives in the fine history of the Hôtel Dieu of Montreal. The other gave up her life and talents to raising the profession of nursing to its modern high level, her powers first being tested against official ignorance, sloth and obduracy which, as a source of suffering and death in the military hospitals of the Crimea differed little from the bestiality of the savages amongst whom Jeanne Mance laboured. The red tape of Scutari was a close collateral of the scarlet war paint of the Iroquois.

Jeanne Mance was one of those who felt the overwhelming desire to devote themselves to religious work which was so evident in France during the first half of the 17th century. She never took the veil, but from her early days apparently was of a deeply religious nature.

Born in Langres, in the Province of Champagne, in 1606, she was a woman of 34 when in 1640 she left her small, quiet town to go to Paris to offer her services in New France. There seems to have been no direct persuasion from her spiritual advisers; indeed, they advised at first against her going. She gave up her sheltered, uneventful life, to submit herself to "unheralded dismays", although she may have known something from the Jesuit *Relations* of the sufferings of missionaries at the hands of the Indians. Sometimes the survivors returned to France, racked, scarred and crippled by their torturers, but willing to offer themselves again in the cause of their religion.

We know little of Jeanne's early life. She was one of 11 children, her family being of the upper middle class, the *bourgeoisie de robe*. Her health apparently was poor, but she had the determination which often overcomes physical frailty. She showed extraordinary resolution and capacity to look after herself in travelling, a young woman unattended, through a countryside of insecurity and violence. This was in France. Later on she appears, solitary and indomitable, journeying up and down the St. Lawrence, and across the Atlantic, under indescribable conditions of discomfort and danger.

In Paris, Jeanne's object was to be assigned some work in New France. Her ability, as well as her religious zeal, attracted the attention of men and women who had conceived the idea of colonizing the Island of Montreal. They had formed La Société de Montréal, whose business it was to collect money and settlers for the colony. One benefactress, Madame de Bullion, a wealthy widow, had set her heart particularly on establishing a hospital in connection with the venture, and soon realized that Jeanne had all the qualities for that part of the work. To her Madame de Bullion entrusted the sum of 20,000 livres as the first donation towards an endowment fund, and later she added further large sums.

The next step in our pilgrim's progress was the port of La Rochelle, where she met Paul de Chomedey de Maisonneuve, 29 years of age, the commander of the expedition. They were to form a perfect team. Maisonneuve was a professional soldier. Jeanne had a steady, orderly mind, with a gift for administration. Both had the same unwavering faith in God, the same impelling devotion to duty, the same selflessness of motive. She was to see her work set well on the path of development; Maisonneuve was to suffer the mortification of being superseded and recalled, his last days being spent in lonely obscurity.

Jeanne's position in the party is not easily defined. She was not a trained nurse. No woman of her social standing did nursing in those days, except as a nun. Her desire to nurse is beyond question, but her work at first was that of administration and management of stores.

There were two ships on the expedition, Jeanne being on one and Maisonneuve on the other. A third boat had left from another port before them. Within two days of leaving La Rochelle (June 1, 1641) they were separated by bad weather, and lost sight of each other for the rest of the voyage. Maisonneuve had to put back to port three times, and lost three of his men, including his surgeon.

The other vessel, with Jeanne on board, had a better trip, but even so only reached Quebec on August 18. Ten weeks on the North Atlantic in a small ship must have made the lower St. Lawrence a very welcome sight to Jeanne. And more pleasant still it must have been to round the Island of Orleans and see Quebec rising before them.

But it was not long before she realized how little the enthusiasm of the supporters of the Montreal venture was shared by the handful of their countrymen struggling to maintain their precarious position on the Quebec cliff. A great rock it was, but the shadows associated with it were neither of the reassuring nature described by the Prophet Isaiah,* nor of the more romantic type so charmingly sketched by Miss Willa Cather. With all its allurement Canada could be a terrible country, and the expedition to Montreal seemed so quixotic, so clearly foredoomed, that the Quebec people must have regarded it with the impatience which foolhardiness always arouses. Besides, they had to watch the procession past their door of men and materials sorely needed by themselves. They were jealous as well, for Montreal would be a rival in trading with the Indians, and she would also be independent, for she would have her own governor. This jealousy extended to Jeanne herself, because she was going to set up in Montreal an order of hospital nuns separate from and independent of the religious order in Quebec.

Jeanne had plenty of time to hear what the Quebec people thought about the expedition, for Maisonneuve did not arrive till more than two weeks later, and we have to imagine her defending her position as well as she could. Maisonneuve put an end to all argument by telling Montmagny, the Governor of Quebec, that he had his orders and was determined to carry them out. He ended with his famous defiance of all the Indians that the woods of Montreal could produce; a challenge which was to be fully accepted.

His firmness cleared the air, and the party spent the winter at Quebec very harmoniously. The Governor and Maisonneuve went up to the Island of Montreal before the winter closed down, and chose a landing place for the follow-

* "As the shadow of a great rock in a weary land." Isaiah, 32:2.

ing year. Jeanne herself made such a favourable impression in Quebec that in addition to being given a comfortable house she gained the support of Madame de la Peltrie, a young and wealthy widow, who insisted on joining the party for the coming spring. There was enough to do, long as the winter was. Boats had to be built and stores collected and packed, which was left largely to Jeanne. She also set herself to learn Iroquois, and to study the ways of the Indians. The church registers at Sillery show her acting as godmother to several baptized Indians.

On May 8, 1642, the party set off up the river. The month of May can be cold on the St. Lawrence, but the weather seems to have been pleasant that year. Progress was slow, and they had every opportunity of observing the river route which was to become so familiar to Jeanne later on. When the Island of Montreal was reached ten days later, the woods were full of flowers and birds, and the party went ashore to set up their altar and tents under the happiest auspices. Ubiquitous as the Iroquois usually were they seem to have had no knowledge of the visitors for several months, and their settling down was not interfered with. They made trips to the mountain, and roamed the woods of the vicinity with a freedom as happy as it was short-lived.

But pleasant as a Montreal summer can be, it never lasts very long, and there is always the winter to be reckoned with. Whatever else their living conditions were like, the settlers must have suffered extreme discomfort from the cold. For the first winter at any rate the whole colony (probably about 50 altogether) seems to have lived in the fort which was built by Maisonneuve as their first defence against the Iroquois. Later on, Jeanne Mance had a house to herself, adjoining the chapel.

The first hospital was a room in Jeanne's house, and although one writer says that there was no sickness amongst the hardy colonists there is evidence that they were not without their illnesses. As Sœur Mondoux suggests,* it is not likely, with all the work required to clear the land of trees, that there were no accidents.

For the first few years the little colony was very busy and very happy. Jeanne looked back on those days as idyllic. She told her chronicler, Sœur Morin, that everyone helped in the common work of the settlement; no doors were ever locked; everybody attended mass before going to work in the morning; "on ne voyait point de péchés publies ni de haines ou rancunes tout n'étoit qu'un cœur en charité, toujours prêts à se servir et à parler des autres avec estime et affection". A simple life indeed; one wonders what recreation they had. There is more than one mention of the singing of chants, but there were probably songs as well. Maisonneuve was a flute player, and may have piped for them.

There is a sad lack of written records by Jeanne Mance herself, especially as regards the first years of the Hôtel Dieu. Amongst its other calamities later on Montreal suffered heavily from fires and in these, the hospital buildings were destroyed on three separate occasions, with many of their records. The Hôtel Dieu today possesses in its archives not a single signature of Jeanne Mance. Her will is preserved in the City archives.

However, there are other sources, the most vivid picture of the first days of the hospital being given by one of its religieuses, Sœur Morin in her *Annales de l'Hôtel Dieu*. She joined the hospital staff in 1662, as a girl of 13, when Jeanne Mance was 56. But the older woman seems to have spoken freely of her experiences, and some of these are recorded by Sœur Morin. Her style is of the simplest, and since she wrote only for her sisterhood, with no idea of its ever being seen by outsiders, her account has great charm.

The other main contemporary account is the *Histoire du Montréal* by Dollier de Casson, the soldier priest, who also came on the scene after the founding of the colony, but was closely associated with Jeanne in her later years.

But nowhere is there any definite description of her person, nor have we any authentic portrait. The *Annales de l'Hôtel Dieu de la Flèche* say of her: "As she was of an attractive enough exterior, and she spoke of God as none could do better, a number of ladies of first quality took a pleasure in seeing and conversing with her". Dollier De Casson, who, from the lively style of his History of Montreal, might have been expected to show a little more interest in her personal characteristics, says only that she had "a captivating and spiritual personality". Abbé Rousseau speaks of "son mérite distingué, ses rares qualités, sa politesse exquise".

But these descriptions still keep us at a distance. It is clear enough that she was an impressive personality, strong-willed, practical, and austere. Whether she had the indefinable quality of being likeable is another matter. One gains the impression that her habit of life was solitary, and that until Marguerite Bourgeoys appeared she seems to have had no intimate friends. Probably there were few if any of her social standing amongst the first colonists in Montreal. There was Maisonneuve of course, but there seems to have been no more than a high mutual respect between them.

Sœur Morin speaks of three main hardships in the life of the founders of the Hôtel Dieu: "Les contradictions; la peur des Iroquois; la pauvreté". The first of these probably caused Jeanne more distress than the other two. It

* Père Vimont's *Relation* of 1643 (quoted by Sœur Mondoux).

included all the opposition which she had met with as soon as landed in the country, and the jealousy which grew as time went on. She managed to maintain her position with dignity, but was under continual pressure from the ecclesiastical authorities at Quebec to yield the control of the Hôtel Dieu at Montreal to them.

Her first most serious problem however, was the gradual weakening of support from France. In 1649 she went down to Quebec and found bad news awaiting her. For various reasons La Société de Montréal seemed to be in danger of breaking up. This was partly ascribed to a growing feeling in France that the position of the Montreal settlement was too dangerous. In any case, the group had almost dissolved. Furthermore, M. de La Dauversière, the manager of the hospital funds, was reported to be bankrupt, and her close friend and adviser, Father Rapine, whose influence with Madame de Bullion was very great, was dead.

It it curious to realize how completely the fate of the Montreal settlement depended on Jeanne at this stage. There was no one to advise her. The Quebec people had no sympathy with her troubles, and there was hardly time to go back to Montreal to consult with Maisonneuve. She may not even have considered doing that, as she seems to have been accustomed to make her own decisions.

At any rate, she sailed for France that October, and succeeded in re-establishing support. Her courage and ability were never more evident. She personified the original spirit of La Société de Montréal, of which Parkman says: "None of the ordinary motives of colonization had part in their design. It owed its conception and birth to religious zeal alone".

It was that zeal which drove Jeanne back to France. There was no question of help from government sources, and if Jeanne had not faced the horrors of another sea journey to encourage the failing enthusiasm of her friends the whole venture would probably have gone to pieces.

She got back to Montreal the following year, with her work well done. But now she found trouble of another kind. The Iroquois were pressing hard. They had evidently determined to destroy Montreal altogether, and with a little more resolution and leadership they could have done so. Jeanne nearly fell into their hands one day, when they captured some of the settlers working in the fields near the hospital. She was alone in the building but some of the workers managed to hold off the attack. One man, named Chiquot, hid himself in the woods during the attack, but was caught and scalped. He managed to escape and was nursed back to recovery in the hospital. Another more determined attack on the hospital in July, 1650, made Maisonneuve decide that it was in too dangerous a position for Jeanne and she was moved back into the fort. The hospital was on slightly higher ground than the fort, in the region now known as Hospital Street, in the heart of the financial district.

Eventually the decision had to be made whether to hold on to the settlement, or to withdraw. Men were being lost in the fighting and were not being replaced. Maisonneuve was willing to fight, but he had to have men, and he had no money to raise fresh forces. At this point Jeanne Mance offered to give him 22,000 livres of hospital capital for collecting recruits in France. In return, the hospital was to receive 100 arpents of cleared land. Maisonneuve readily accepted the offer. He said that the situation was so bad that if he could not get together at least another 100 men he would not come back to Canada at all, but would regard the whole venture as a failure; and from a soldier's point of view he was quite right.

Maisonneuve sailed from Quebec on November 5, 1651, and there must have been fervent prayers for his return. The soonest he could be expected back was in the following summer, and he might easily not come that year at all. The winter passed, and in the spring Jeanne persuaded the governor to let her go down to Quebec for advance news. But all she learnt was that Maisonneuve would not be back for another year. However, his letters to her said that he was collecting men and money. Madame de Bullion, although always keeping herself in the background, had again opened her purse. There was therefore nothing for Jeanne to do but go back to another winter in Montreal—and the savages.

In the spring of 1653, as soon as the ice was out of the river she went down to Quebec again, nearly falling into the hands of the Indians on the way. She got word that at last Maisonneuve was on his way, and sent cheering messages back to Montreal, but her messengers were held up by Indian roving bands and came back with the depressing news that Three Rivers was blockaded.

However, Maisonneuve arrived at last, although not until September 22. He brought his 100 men, and probably few ships have ever sailed up the St. Lawrence with more sorely needed help. It was a transfusion for Montreal at a gravely critical stage of collapse. Maisonneuve had had his own difficulties. He had managed to collect a force of soldiers and settlers, "hommes fort et courageux". But, as M. Massicotte reminds us, the colonization expeditions of the 17th century all had one point in common; the ships were always leaky, and usually unhealthy. It was common for them to have to put back to port for repairs, as had happened on Maisonneuve's first voyage. On this later trip the boat was leaking badly enough when they started. They ran into bad weather and had to go back again, barely mak-

ing the harbour in time. The recruits were both frightened and enraged, and Maisonneuve did not dare to let them land, whilst another ship was being got ready, but kept them on an island offshore. Even so, a few seem to have swum ashore rather than undertake the voyage. Food ran short, and eight men fell ill and died. When at last they reached Quebec the ship ran on to a reef and could not be refloated. It was left there and burnt.

Maisonneuve had done better than Jeanne had hoped. He brought with him, from his own town of Troyes, Mademoiselle Marguerite Bourgeoys, who was to fill so large a place in the educational life of Montreal. She was 14 years younger than Jeanne, but they soon became fast friends. They spoke the same spiritual language, and were of the same social standing. Maisonneuve had made no mistake.

But Jeanne was getting older, and needed help in her hospital work. The original plan of sending out nursing sisters from France had never been carried out, and no one seemed to be concerning themselves very much about the matter. In 1657, however, Jeanne broke her arm in a fall on the ice, and the injury disabled her for the next two years, eventually forcing her to go back to France to seek help.

In 1659 she brought back with her the three hospitalières of the Order of St. Joseph de La Flèche, who formed the devoted original nursing staff of the Hôtel Dieu. From then on they did the actual nursing. They were a fine trio: Judith Moreau de Brésolles; Catharine Macé; and Marie Maillet. They early learnt what it was to follow Jeanne Mance, both on the terrible journey across the Atlantic, and later in Montreal. Even the veteran traveller Jeanne permitted herself some comments on this particular trip. The vessel was a so-called hospital ship: it had been used in the navy for the sick and had never been properly cleaned; also as usual, it was leaky. Sickness developed on board almost at once, and about 18 of the passengers died at sea. Most of the women were very ill, Jeanne and Marguerite Bourgeoys among them, but the nursing of the sick had still to be carried on. The trip lasted 71 days. Jeanne speaks of their relief on coming into the Gulf and of being able to obtain fresh drinking water. The water on the ship had been rationed, and, as she described it: "c'était méchante et quelquefois pleine de vers,* ce qui était assez ordinaire sur la mer; ceux qui en ont fait le trajet n'en seront pas estonnés".

And what were they coming to? By the time the nurses reached Montreal it was October, and they went ashore to the little huddle of houses amongst charred stumps and half cleared fields, to begin their work. Desolate enough Montreal must have looked then, but the inhabitants gave them a welcome that made up for all their tribulations.

The actual nursing in the Hôtel Dieu, especially when Jeanne was alone in the work is not described. But there is an account of the hospital itself. The building was half wood and half stone. The men's ward contained six beds and the women's two. There was a large fireplace at the end of the men's section which did for both wards. All the cooking was done in this fireplace, and the apothecary had a corner there for work.* Here also the linen and dressings were washed, "ce que arrivait assez souvent, ayant peu de linge et bien des blessés". The clothes were dried in the attic, off which there was another very small room, about 6′ x 9′, in which the sisters lived.

Fig. 1

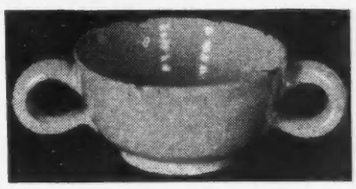

Fig. 2

Fig. 1.—One of the original druggist's jars in the pharmacy of the Hôtel Dieu.

Fig. 2.—A 17th century hospital feeding cup used at the Hôtel Dieu.

(Kindness of Sœur Mondoux, Hôtel Dieu, Montreal.)

The ward opened into the chapel, so that the patients could take part in the services, but sometimes the noise of the people coming and going disturbed the sick. There was a small room at the foot of the chapel where the sisters made their retreat. It was without light or ventilation, and Sœur Morin says she was unable to complete the allotted time of her retreat there as it made her ill.

Jeanne Mance had separate rooms from the hospital, but must have shared much of the common hardship of cold. Sœur Morin speaks of the snow drifting into the hospital through the cracks in the walls, so that the first duty of the sisters on winter mornings was to sweep drifts out of the rooms. In really cold weather most of their food froze hard, the bread, the soup, and the vegetables.

The menu was not a varied one. For years they had practically nothing but corn, lard, peas and beans, "l'anguille salée", and pumpkins. For ten years, Sœur Morin says, she saw

* This could be almost literally translated by Kipling's description of the water brought by Gunga Din: "it was crawlin' and it stunk".

* Sœur Brésolles was the apothecary for years, and gave medical advice and treatment as well. Her skill became famous. Sœur Morin says. "Enfin les madades croyois ne pouvoir mourir quand ils s'étois mis entre ses mains ou gouverné par ses conseils. Ce qui passa à un excès à faire rire."

no fruit except some wild plums. Strawberries and raspberries could be picked in the fields, but only at the risk of losing one's scalp. Sometimes Jeanne would send them in some fish or game. It had not taken the settlers long to appreciate moose meat. Sœur Morin has a good description of the moose, and of its inconvenient tendency to attack hunters and trample them in the snow.

The sisters truly lived in poverty, for amongst their "contradictions" had been the loss through M. de la Dauversière of a large part of their original endowment. Their very dresses were so worn and patched that the original material was hardly recognizable. One day Maisonneuve, going through the hospital with Jeanne Mance and some visitors, began to chaff the nurses about the variety of repairs to their dresses. As usual, he was able to make them laugh at themselves, and soon the whole group was amusing itself with jokes about how each garment had been renewed with various pieces of cloth. Jeanne was able to identify one piece as belonging to an old dress she had passed on to them, and Madame d'Ailleboust thought she recognized something of hers, and so on; Maisonneuve highly enjoyed himself all the time. The whole lighthearted scene is delightfully described by Sœur Morin.

As this was the only hospital west of Quebec it had a wide territory to draw from. Frequently there were Indian patients, either friendly Hurons or even wounded Iroquois themselves. Some of these latter responded to kindness, others did not. Sœur de Brésolles one day was pushed into a cupboard by an Iroquois patient who tried to stifle her by shutting the door on her. The other patients went to her help, but when they pulled him off he laughed uproariously and said he had only been joking.

The new hospital staff soon began to experience the terror of the Indian attacks, and ungovernable fear would sometimes break down their self-control. Sœur Morin herself seems to have been the coolest, perhaps because of her having been born and brought up in Quebec. When the tocsin sounded Sœur Maillet fainted and Sœur Mace hid in the sanctuary, unable to speak or move. Sœur Morin says: "Moi, qui savoit le lieu de leur retraite, je les allois consoler, aussy tot que j'avais appris que les Yroquois s'étoit retirés". Sœur de Brésolles was terrified too, but was able to go on with her work, and she and Sœur Morin would go up into the tower to keep the bell going. From there they sometimes were able to see the actual fighting, in which even the women sometimes took part.

There is little recorded of Jeanne Mance's last years, except that her health gradually failed, confining her to bed for long periods. In 1662 she had made yet one more journey to France (her third). It was her melancholy duty to act as Maisonneuve's representative at the handing over control of the Montreal settlement to the Order of St. Sulpice, since the Company of Montreal had broken up. A year after she returned Maisonneuve was removed from his post as Governor of Montreal, and was recalled to France. It was a saddening close to their association. Sœur Morin has some charming little notes on Maisonneuve. In one place she says: "Mais je reviens à M. de Maisonneuve dont je me suis un peu divertie. il etait sans pareil en constance dans l'adversité. Ce qui auroit attristé un autre, ou mis en colère, ne faisait que le faire rire et mieux

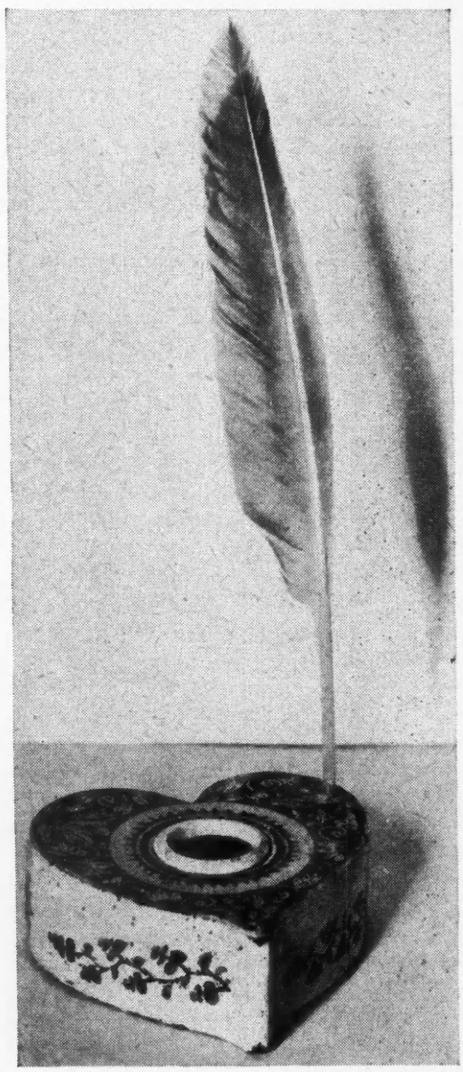

Fig. 3.—Inkstand, probably belonging to Jeanne Mance.
(Kindness of Sœur Mondoux, Hôtel Dieu, Montreal.)

divertir, trouvant des avantages à ce qu'il disoit, dans ses disgraces, qu'on ne savoit pas. Quand il avoit des sujets de chagrin il rendit visite à ma Sr. de Brésolles ou à la Sœur Bourgeois, afin de rire de plaisir; elles riaient aussi avec lui et luy montrois grande joie de ses peines, ce qu'il aimoit beaucoup."

In June, 1672, Jeanne gathered enough strength to attend the laying of the foundation stone of the new Notre Dame parish church, which was to become the present Notre Dame Cathedral. She was one of the five chosen to have their names inscribed on leaden plaques let into the foundations. Who more than she deserved the honour? She had known hard, grim years, but there had been light as well as shade, and she had never lost faith. She had not only established her hospital but had helped to preserve the settlement itself through soul-shaking days, by her cool head, her courage, and her strength of character.

She died June 18, 1673, and was buried in the chapel of her hospital.

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ASSOCIATION NOTES

Short-Wave Diathermy

In the April and June issues of the *Journal*, new regulations of the Department of Transport, Radio Division, for the control of radio interference from short-wave diathermy machines, were published. The essence of these regulations is that by January 1, 1948, short-wave diathermy machines must be shielded adequately or replaced by machines which have controlled radio frequencies.

It has come to our attention that a persistent rumour is circulating in Canada to the effect that the Department of Transport does not intend to enforce the regulations and that physicians may continue to use unshielded and uncontrolled short-wave diathermy machines for a further period of years. Enquiries addressed to the Controller of Radio, Department of Transport have elicited the information that this rumour is without foundation and "ALL EXCESSIVE RADIATION ON COMMUNICATION FREQUENCIES MUST BE SUPPRESSED ON OR BEFORE JANUARY 1, 1948. THIS DEPARTMENT DOES NOT CONTEMPLATE EXTENDING THE ABOVE DATE FOR SUPPRESSION OF EXCESSIVE RADIATION."

The Federal Communication Commission in the United States has announced that short-wave diathermy equipment manufactured prior to 1947 may continue to be used in that country for a period of five years, provided that no interference is caused to an authorized radio service. This announcement has no effect in Canada where conditions effecting radio communications are quite different, and where it is proposed to eliminate radio interference by general rather than individual action.

The attention of all Canadian doctors is again called to the necessity of taking steps to suppress radio interference from electro-medical apparatus by January 1, 1948.

MEDICAL SOCIETIES

Brant County Medical Association

The May meeting of the Brant County Medical Association was held in the Willett Memorial Hospital, Paris, Ontario, on May 29. Dr. Ian McDonald of Christie Street Hospital, Toronto, gave an address on "Medical Aspects of Thyroid Disease".

The B.C. Surgical Society

The British Columbia Surgical Society, incorporated under the Societies' Act has now commenced to function. The executice consists of: *President*—Dr. Lyon H. Appleby; *Vice-president*—Dr. T. H. Lennie; *Secretary-treasurer*—Dr. Roy Huggard; *Directors*—Dr. A. B. Schinbein, Vancouver; Dr. A. C. Frost, Vancouver; Dr. Lee Smith, Vancouver; Dr. S. A. Wallace, Kamloops; Dr. G. C. Kenning, Victoria.

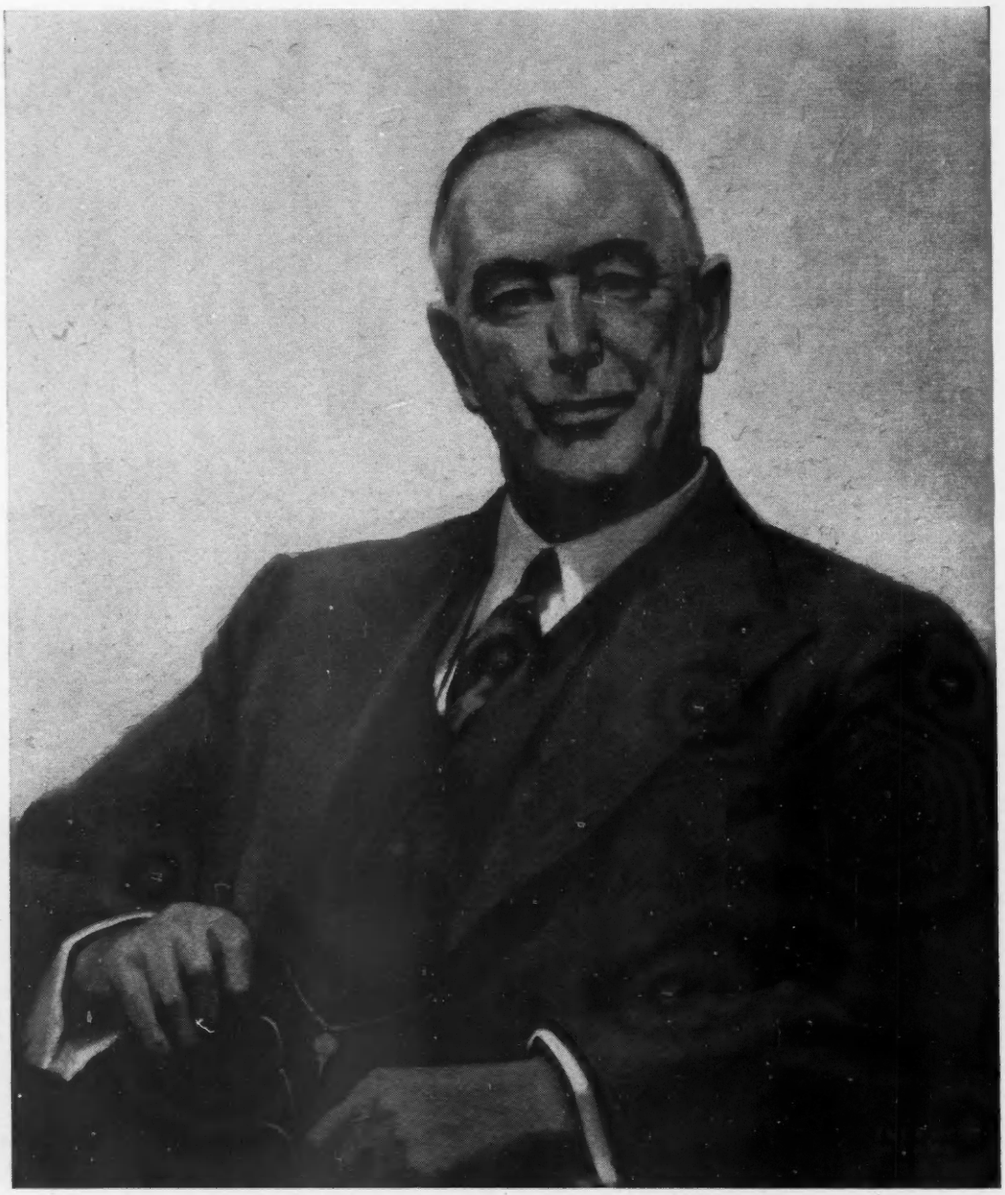
Canadian Anæsthetists Society Quebec Division

At the last meeting of the Canadian Anæsthetists Society, Quebec Division, held on May 3, 1947, the following members have been elected to fill the following charges. *Representatives at the National Council of the Canadian Anæsthetists Society*: the President of the Quebec Division, R. Rochette; Harold Griffith; George Cousineau. *To the Executive of the Canadian Anæsthetists Society, Quebec Division*: *President*—R. Rochette; *Vice-president*—F. H. Wilkinson; *Secretary-treasurer* — M. Clermont; *Assistant Secretary-treasurer* — J. Beaudry.

The Montreal Medico-Chirurgical Society

The annual meeting of this society was held on May 16. After the routine business the retiring president, Dr. Lorne C. Montgomery, in his presidential address gave a short review of the work of the Society for the season. There had been an increase in membership and the large attendance at the meetings had been most encouraging. The success of the annual fall clinical convention was particularly striking, the attendance at the hospitals being close to 400.

Dr. Montgomery felt that he might fittingly refer to the whole-hearted welcome which he and all Canadian medical officers had experienced in Great Britain during the war years. In addition to his acknowledgment of this unwearying hospitality he had been greatly impressed with the resolute tenacity with which medical society meetings had been carried on, under physical difficulties which might well have been taken as an excuse for their discontinuance, but which were never



THOMAS CLARENCE ROUTLEY, M.D., C.B.E., LL.D., F.R.C.P.[C.]

At the Sixty-seventh Annual Meeting of the Ontario Medical Association held in Toronto, May 12 to 16, 1947, the unique distinction of Honorary Membership was conferred on Dr. T. C. Routley. After referring to the outstanding services of Dr. Routley to the Canadian Medical Association and to the Ontario Division, the President, Dr. William Magner, paid tribute to his more recent activities in the field of international relations. To mark the occasion, the Ontario Medical Association presented the new Honorary Member with his portrait, which is reproduced herewith. The portrait is the work of Mr. Cleeve Horne, O.S.A.

allowed to affect the customary high standard of work produced.

The evening closed with a most pleasing demonstration by Dr. Fred. J. Burgess of his hobby of nature studies in colour photography. A long series of slides was shown of many varieties of mushrooms and wild-flowers, and the artistic quality of arrangement and lighting produced delightful effects.

Physiological Society of University of Toronto

Professor M. A. Van Bouwdijk Bastiaanse, Professor of Obstetrics, University of Amsterdam, addressed the Physiological Society of the University of Toronto recently on "The Kidney in Pregnancy". He was the medical leader of the Netherlands Resistance Group. Dr. H. B. VanWyck, Professor of Obstetrics and Gynæcology, introduced him in the Dutch language. Professor Bastiaanse described his experiment on dogs in which he constricted the blood vessels to the uterus. This resulted in a rise in blood pressure.

In eclampsia cases, blood plasma albumen is reduced. The albumen content of the blood is the guide to the amount of protein given in the diet. He uses milk to make a high protein diet and can give up to 250 grams of milk curd daily. He advised against giving sodium. Its use may result in constriction of all the small blood vessels in the body. Prognosis is worse in cases where retinitis occurs. He stated emphatically that convulsions in pregnant patients in the absence of hypertension are not due to eclampsia but to some other cause.

LILLIAN A. CHASE

Saint John Medical Society

Dr. Robert Gregory, assistant superintendent of the Provincial Hospital at Fairville, was the speaker at the monthly meeting of the Saint John Medical Society in May. His topic was "Sciatica and Brachial Neuritis". The presentation was followed by brisk discussion, particularly on the part played in these syndromes by hypertrophic changes and disc protrusions. The speaker covered the various nerve manifestations in the two groups caused by systemic disease.

The following officers for 1947-48 were elected by the Saint John Medical Society at their Annual Meeting in May. *President*—Dr. F. R. Connell; *Vice-president*—Dr. Stephen Clark; *Treasurer*—Dr. Robert Gregory; *Secretary*—Dr. George White; *Executive Members*—Dr. V. D. Davidson; Dr. W. D. Miller; Dr. F. K. Stuart.

A. S. KIRKLAND

Winnipeg Medical Society

The regular monthly meeting of the Winnipeg Medical Society was held in the Medical College on April 18. Dr. A. W. Trueman, President of the University of Manitoba, spoke on the place of the doctor in society. He stated that the physician was rarely depicted in his true light in fiction, the usual treatment being over harsh or over sentimental. One of the clearest pictures, he said, was that of the doctor in Jane Austen's "Emma". He never appeared, but one got a fine appreciation of the man from the remarks of the various characters.

Dr. L. R. Coke spoke of Current Methods in Treatment of Heart Disease, especially in the Massachusetts General Hospital, Boston.

Central Medical Society

A meeting of the Portage la Prairie and District Medical Society was held on April 22 at the Hotel Portage. The meeting was well attended by all of the medical profession residing in the city and rural municipality of Portage la Prairie, as well as Dr. Jubb from MacGregor and Dr. Ivens from Carberry. The special guests of the evening were Dr. A. E. Childe, Dr. Bruce Chown and Dr. M. T. Macfarland, all of Winnipeg.

Following dinner Dr. Chown gave a very interesting address on the Rh factor, outlining methods of sensitization of Rh negative individuals and the possible results of such sensitization. He showed coloured lantern slides illustrating clinical forms of erythroblastosis fœtalis and outlining the treatment now being used. Dr. A. E. Childe spoke to the gathering on the subject of protruded intervertebral discs. His address was profusely illustrated with lantern slides demonstrating the technique used and reproductions of x-ray films illustrating a great variety of protrusions both in the lumbar and cervical regions.

Dr. Macfarland, executive secretary of the Manitoba Medical Association very briefly outlined some of the work being carried on at the present time by the Executive of the Association and a few of the problems with which the Association is faced.

ROSS MITCHELL

La société médicale des hôpitaux universitaires de Québec

Séance de la société médicale des hôpitaux universitaires de Québec, le 11 avril 1947.

HERNIE INTERCOSTALE PULMONAIRE.—J.-P. Roger et J.-M. Lemieux.

Ces deux auteurs ont observé un cas de hernie intercostale dans la partie antérieure du 3ème espace intercostal droit à la suite d'un traumatisme. La travail fait une revue de la pathologie de la lésion et des procédés chirurgicaux dont on dispose pour traiter ces hernies. Leur technique a consisté à fermer la brèche à l'aide de lambeaux périostés détachés des côtes voisines.

TUBERCULOSE EXPERIMENTALE DU HAMSTER DORÉ (Cricetus auratus).—Maurice Giroux.

L'expérience rapportée avait pour but de connaitre la réceptivité d'un nouvel animal de laboratoire, le hamster doré, vis-à-vis des souches de bacilles tuberculeux humains d'une part, et bovins d'autre part. Cet animal s'est révélé peu sensible à l'inoculation de bacilles tuberculeux humains. Cependant, il est beaucoup plus rapidement et fortement tuberculisé par les souches bovines.

CINQ OBSERVATIONS DE CANCER BRONCHOGÈNE.—Louis Rousseau, Maurice Giroux, Jules Hallé.

Observations de 5 sujets atteints de cancer bronchogène dont l'âge moyen est de 48.4 ans. Chez trois malades, le cancer bronchique était masqué par un syndrome de suppuration pulmonaire; chez un autre existait une association tuberculeuse. La tenacité des hémorragies prend une valeur pathognomonique dans certaines formes de cancer bronchique. La situation haute de ces tumeurs bronchiques fait que leur diagnostic est facile par exploration bronchoscopique mais elle s'oppose à tout traitement chirurgical efficace. Des cinq cas observés, tous étaient morts 8 mois après l'établissement du diagnostic histopathologique.

DYSEMBRYOME THORACIQUE ET CHORIO-ÉPITHÉLIOME CHEZ UN MALADE DE 12 ANS.—Maurice Giroux et Roland Desmeules.

L'observation rapportée est celle d'un jeune patient de 12 ans, ayant présenté un syndrôme consistant en douleurs thoraciques, toux et surtout dyspnée, qui a évolué fatalement en deux mois. La nécropsie révèle qu'il s'agit d'un dysembryome du poumon et médiastin, avec chorio-épithéliome des poumons, foie et rein. La pathogénie de ces tumeurs dysembryoplasiques est discutée en rapport avec leur évolution chez ce malade.

Séance de la société médicale des hôpitaux universitaires de Québec, le 25 avril 1947.

MALADIE DE STEINERT.—H. Pichette et J. Audet.

Les auteurs rapportent l'observation d'un cas de la maladie héréditaire et familiale décrite par Steinert. Rencontrée chez une jeune femme de 29 ans, cette pathogénie a été caractérisée: 1° par de l'affaiblissement des muscles et de l'atrophie musculaire. 2° par de la myotonie ou augmentation du tonus musculaire. 3° par une cataracte bilatérale. On a noté une diminution du métabolisme basal et une cholestérinémie élevée. A part l'absence de certains troubles trophiques, la symptomatologie de cette maladie était bien celle dépeinte par Steinert et Curschmann.

AFFECTIONS URINAIRES CHIRURGICALES AU COURS DE LA GROSSESSE.—J.-N. LaVergne et A. Mercier.

La néphrectomie, au cours de la grossesse a les mêmes indications qu'en dehors de cet état. La grossesse n'en est pas une contre-indication. Chez une malade néphrectomisée dans le cours de sa grossesse, la gestation suit le cours normal de son évolution. La durée du travail n'est pas prolongée, les suites de couches demeurent normales. La tuberculose rénale unilatérale et le cancer du rein commandent l'intervention pendant la grossesse. L'avortement ou l'accouchement prématuré ne sont pas indiqués. L'hydronéphrose chez les femmes enceintes n'exige pas l'intervention immédiate. Une surveillance étroite, le drainage des bassinets peuvent, dans bien des cas, obvier à l'intervention. La maladie polykystique des reins chez la femme enceinte demande pour l'obstétricien une surveillance particulièrement attentive. Le meilleur temps pour intervenir, chez les patientes enceintes, porteuses de calculs urinaires, est dans la période du post-partum. Une grossesse sera permise aux patientes néphrectomisées si on a la certitude de l'intégrité du rein restant. Dans les cas de tuberculose rénale opérée, si l'autre rein est certifié sain, les auteurs recommandent une attente de deux à trois ans.

LE CYCLOPROPANE.—A. Paquet et L. Rinfret.

Les auteurs étudient brièvement la composition chimique, la pharmacologie, le mode d'emploi, les indications et les contre-indications du cyclopropane. Ils rapportent les résultats qu'ils ont obtenus de l'usage de cet anesthésique chez plus de 2,000 malades, soit seul, soit associé à d'autres anesthésiques. Ils passent en revue les complications que peuvent survenir au cours et après l'administration du cyclopropane et indiquent les moyens thérapeutiques à prendre, le cas échéant, afin de les éviter dans la mesure du possible ou d'y remédier lorsqu'elles se sont installées.

DIAGNOSTIC DIFFICILE D'UN ÉTAT INFECTIEUX CONSECUTIF À UN AVORTEMENT PROVOQUE.—Fabien Gagnon, Renaud Lemieux et H. de St-Victor.

Une jeune fille de 26 ans fait une infection puerpérale à la suite de manœuvres abortives accomplies dans les conditions les plus douteuses. Une thérapeutique anti-infectieuse comportant sulfamidés, pénicilline, streptomycine, petites transfusions de sang, est mise en action dès les premiers jours de l'apparition des troubles. Cette malade semblait n'être porteuse d'aucune lésion cardiaque à ce moment et rien, dans l'histoire antérieure, ne peut faire croire à une atteinte du cœur. Après une période infectieuse aigue manifestée par un état de fièvre assez élevée, une rémission des troubles se fait et tout semble près de rentrer dans l'ordre. On réalise à ce moment que sur cet état infectieux puerpéral, s'installe une infection rhumatismale cardiaque. La courbe thermique, qui est plutôt régulièrement haute au début, lors du syndrome puerpéral, et qui est modifiée par les antibiotiques, change de caractère et devient à oscillations de grande amplitude lors du syndrome rhumatismal. L'inaction des anti-biotiques à ce moment, l'action manifeste du salycilate, la constatation de lésions typiques au niveau du cœur, conduisent à affirmer le diagnostic de rhumatisme cardiaque consécutif à une infection puerpérale.

Winnipeg Medical Society

The annual meeting of the Winnipeg Medical Society was held in the Medical College on May 16. The committee on membership reported that approximately 500 members were enrolled. The retiring president, Dr. W. F. Tisdale, gave an interesting address on Nature Study as a Hobby, illustrated with slides of many wild birds, some species of which are extinct or in danger of extinction.

The election of officers resulted as follows: *President*—Dr. C. E. Corrigan; *Vice-president*—Dr. R. A. Macpherson; *Secretary*—Dr. K. A. Trueman; *Treasurer*—Dr. Sam Boyd; *Trustee*—Dr. Marjory Bennett.

Vancouver Medical Association

The annual meeting of the Vancouver Medical Association was held on May 6, and the Election of Officers for the ensuing year took place at this time. The officers elected were: *President*—Dr. Geo. A. Davidson; *Vice-president*—Dr. Gordon Johnston; *Secretary*—Dr. W. J. Dorrance; *Treasurer*—Dr. Goron Burke; *Delegates at large*—Drs. Henry Scott and Roy Huggard; *Editor*—Dr. J. H. MacDermot; *Elected to Board of Trustees*—Dr. A. C. Frost.

Following the meeting, Dr. A. E. Graner, Ph.D., President of the B.C. Electric, who had been invited as guest speaker, addressed the audience, taking as his subject, "The Direction in Which We Are Going".

Dr. Graner, who is a graduate of the University of British Columbia, is a well-known speaker on economic questions, and his remarks received the closest attention.

CORRESPONDENCE

Fractures of the Mandible

To the Editor:

It was with some interest but more surprise that I read Dr. Gerrie's criticism of the above article appearing in the March edition of your *Journal*. One might have thought that a man with such a vast experience in this particular subject would have acquired more realism and less idealism in his views on fractures of the mandible.

I believe he is unduly alarmed, and is taking too much for granted when speaking of catastrophies. This procedure had been carefully considered and well planned and a catastrophe was neither expected nor feared. The case in question had absolutely no suitable teeth for wiring and the procedure undertaken involved no more risk than his direct wiring or pinning. Dr. Gerrie speaks of this area as the only one having a ready and waiting perfect anatomical splint. To this I would say that there are many such areas in the body, *e.g.*, tibia and fibula, radius and ulna, metatarsals and metacarpals, but the presence of such perfect readily available splints does not in any way guarantee either perfect opposition or immobilization.

If there is fiction in what I have written then I suggest that he read fiction, as fiction is often more revealing than fact.

It may be true that general anæsthesia is not indicated but one does not need to be a master observer to see that it is generally given outside of large centres. When Dr. Gerrie speaks of wiring fractures under small doses of morphine and says that it is not painful I believe he is disregarding obvious facts. He has admitted that wiring might be tedious but minimizes the time factor. The one-half hour might well be true for his excellent clinic and for other large, well equipped, well staffed clinics but it certainly is not true for the general run of general surgeons, and I use the term general in the broadest sense of the word. I venture to say that secondary immobilization by cross wires and

elastics can also be difficult and painful and I have known more than one patient who absolutely refused to have it completed without anæsthesia.

I presume Dr. Gerrie's chief worry is about infection and that also was mine. However, if one realizes that the mandible is covered with periosteum and a densely adherent mucous membrane communicating with a continuously infected cavity it must of necessity be frequently inoculated with disease-producing bacteria. The mandible through the alveolar sockets is also repeatedly inoculated yet one must agree that this bone is relatively immune to infection. Infection certainly does occur, but it is more frequently well localized and not extensive, and heals without gross destruction of bone. In fact, I believe that the mandible is the most resistant bone in the body, and certainly bone plating carries but little more risk than direct wiring and gives a well apposed mobile jaw.

I certainly did not intend to advocate plating in every fractured mandible but said "at least in selected cases", and I still believe this procedure to be sound. The anatomic area is definitely not dangerous. There are, of course, a few important structures in the area but every good surgeon identifies and protects these structures on exposure and thinks little about them after isolation.

Bone plating gives accurate apposition and good immobilization and a mobile open jaw is a tremendous advantage, as was shown in the case described. I still believe the "common method in use today" has much to be desired, and what I have said about it I believe to be true in spite of the criticism by a very competent man. If in his desperation Dr. Gerrie condescends to use the bone plate, I believe he may find his salvation.

J. D. McINNES.

Sudbury, Ontario,
May 13, 1947.

A Testimonial Withheld

To the Editor:

We have all had our doubts about testimonials relating to the efficacy of different drugs, etc.

I have recently had a first hand account of the value of raz-mah which is advertised for asthma. During a conversation with our local undertaker who is an asthmatic, I asked him what he was doing for his condition and he told me the usual story of trying everything that was being advertised. He happened to see the raz-mah ad and he was going to try it, until he noticed glowing testimonials from two individuals whom he buried two years previously.

A. SEARLE.

Flin Flon, Man.,
May 19, 1947.

SPECIAL CORRESPONDENCE

The London Letter

(*From our own correspondent*)

NEW LIGHT ON MORBIDITY

Since the autumn of 1943 a regular series of surveys has been made of the incidence of ill-health in random samples of the population, and a report has just been issued reviewing the findings of the survey up to the end of 1945. The evidence thus provided at last removes a notable defect in vital statistics which until now have never provided any information concerning the amount of non-fatal illness in the community. Whilst a much longer period of observation is necessary before any definite conclusion can be drawn, several interesting features have already emerged from these surveys which, it is intended, will remain, and probably will be developed, as a permanent feature of our future health organization. For instance, the highest incidence of illness in 1945 was in March (82%), falling to 69% (the lowest figure) in September and October, and in the same year one out of five people had "a cold" in January compared with only one out of twenty in July. The amount of new illness did not increase with age, and the liability to injury was greater under 30 years of age than it was over this age. The annual loss of men's work through illness was about 230,000,000 men-days, and the estimated average number of days incapacity caused by all sickness, in the winter of 1944-45, was 1.4 per person per month.

THE NUTRITIONAL TANGLE

A recent provocatively extravagant article in the medical press has served to bring to the surface an undercurrent of dissatisfaction concerning the present nutritional status of the country. How much of this dissatisfaction is justified, it is difficult to say, principally because of the lack of reliable information, a lack for which the Government must accept responsibility. Nutrition and politics make uneasy bedfellows, but it will be generally deplored if the former becomes a subject of political controversy.

One of the major difficulties in assessing the present situation is the reluctance of the authorities to divulge figures which they possess concerning the consumption of food by representative families. The only information forthcoming is the total amount of foodstuffs going into consumption—a figure of little scientific value. Whilst there is no doubt that certain sections of the community, such as expectant and nursing mothers and young children, are receiving adequate supplies of food, there is no corresponding certainty concerning other sections of the community.

Particularly worrying is the problem of the adolescents. No definite figures have been published to justify a dogmatic statement, but there is a growing censensus that the adolescent is not receiving adequate calories, with the results that the average weight of these youngsters is tending to fall. This is a problem which has been accentuated by the rationing of bread. So long as bread was freely available it was possible for the adolescent to obtain all the calories he required. Today this is not always the case. The possibility of such an eventuality was probably one of the main reasons for the postponement of bread rationing until the date when it was actually introduced. It is to be hoped that reliable figures will soon be available to confirm, or disprove, this most disquieting feature of the post-war era.

HEALTH OR WEALTH?

A disturbing feature of the drive for increased production in industry is revealed in a report on Day Nurseries, published in the *British Medical Journal*. This report is based upon a particularly careful survey of over a thousand children carried out by one observer over a period of a year, and it includes repeated examinations of the children. The results show that the incidence of respiratory tract infection was from two to eight times greater in the nursery children than among those living at home, and that a significantly higher incidence of specific infectious fevers occurred among the nursery children. Most significant of all, perhaps, is the observation that among the children under the age of two years the average weight was considerably less than that of children living at home and that their general condition was inferior.

These figures may not be surprising to pædiatricians but they do seem to emphasize the possible dangers involved in the present demand for women, both single and married, to return to industry. Exports are undoubtedly essential to the survival of the country, but the question is now being asked as to whether the health of the future citizens of the country is a price that can be afforded for increased exports. Planning without forethought and knowledge is a two-edged weapon, and it is to be hoped that the planners of today will take cognizance of the writing on the wall.

PATENT MEDICINES

The Pharmaceutical Society has just presented to the Ministry of Health a report based upon a five-years' survey of the problem of patent medicines. Claiming that little advance has been made since the now famous Select Committee which reported on the subject in 1914, the Society puts forward a series of recommendations aimed at controlling the trade in proprietary medicines. These include the suggestion that it should be the statutory duty of the Ministry of Health, advised by an expert committee, to control the standards and advertising of proprietary medicines. These standards are to include requirements for the disclosure of composition in approved words and quantities, and false, misleading or exaggerated claims are to be prohibited. A register of medicines and manufacturers, it is recommended, should be kept, the sale of unregistered medicines being prohibited.

Whether this new move will be any more successful than previous ones remains to be seen, but there can be little doubt that the increasing health-consciousness of the people will lend itself to further abuses in this line unless action is taken in the near future. Whether Government action will be forthcoming depends largely upon the popular demand for action, and unfortunately such demand has never yet been sufficient to encourage the authorities to take adequate action. On the other hand the new vested interest that the Ministry of Health is acquiring in the health of the country after next April may act as an efficient stimulant to action.

WILLIAM A. R. THOMSON

London, June, 1947.

The Holland Letter

(From our own correspondent in Holland)

TEMPERATURE, PULSE AND RESPIRATION IN MALARIA

A study of temperature, pulse and respiration curves of 130 patients suffering from quartan malaria (mostly chronic) and of the curves of many more patients suffering from tertian malaria and subtertian malaria by Dr. E. van der Kuyp, government physician at Willemstad, showed that the course of respiration and pulse showed more constantly and more clearly the quartan, tertian of subtertian type than the temperature curve. Often the three curves supplemented each other in such a way as to make the type of the fever very clear. The pulse curve sometimes showed the existence of a duplex or triplex quartan infection; the symptom of anticipation and postponement of the fever could be recognized.

During the treatment, the temperature was the first to become normal. Pulse and respiration curves went on showing slight but unmistakable elevations and became at last normal too. If pulse and respiration curves did not become normal, but went on showing the type of infection quite clearly, a relapse followed. At the end of the incubation period pulse and respiration curves sometimes heralded the coming attack by typical variations.

If hourly readings are taken, the observations mentioned above are even more pronounced. The characteristic course makes it probable, that the above mentioned symptoms are due to a schizogony on a small scale. The quantity of liberated toxins is too small to influence the temperature, but large enough to influence the pulse and respiration rate, these being more sensitive to these toxins.

EXCERPTA MEDICA

Three professors of the Amsterdam University have founded *Excerpta Medica*, a new international review, published monthly in the English language. Prof. M. W. Woerdeman, head of the department of anatomy and embryology of the Amsterdam University, Prof. W. P. C. Zeeman, famous ophthalmologist, Prof. A. P. H. A. de Kleyn, director of the clinics for oto- rhino- and laryngology of the Amsterdam University, will publish under their direction monthly an English review of the whole medical science. About 6,000 medical papers and journals are regularly read and analyzed by the 400 editors and 3,000 correspondents in the world. With the help of the Russian ambassador in the Hague, even Russian scientists can contribute to the new medical review. Prof. Tron from Moscow and Prof. Filatov from Odessa will publish regularly in this new international medical journal of reviews and analyses of the medical science.

Excerpta Medica will try to be a complete monthly abstracting service of the world medical literature, comprising 15 sections and covering the whole field of theoretical and clinical medicine. It will be published in 15 separate volumes. Section XIII (Dermatology and Venereology) is just published and comprises 289 analyses and summaries. Informative abstracts for readers, who want to get an idea about the whole literature on a special subject, without having to spend time on the whole literature on the subject, are published in every volume.

The Dutch Government has given all possible help to the publication of this important Dutch medical work.

A NEW VITAMIN IN BUTTER

In the laboratories for physiological chemistry of the University of Amsterdam under direction of Prof. B. C. P. Jansen who was the first to compress vitamin B into pure crystals, researches have been going on during the last years on the favourable influence of butterfat on the growth of young rats. Prof. Jansen and Dr. J. Boer found in the first place, that summer butterfat showed a more favourable influence on the growth of young rats than other fats, even winter butterfat. The factor responsible for this action is not identical with one of the known vitamins. (The glycerides of the fatty acids out of summer butter, have, if the unsaponifiable fraction from summer butter is added, a growth-promoting action, equal to that of summer butter.)

The factor responsible for this better growth apparently is an unsaturated fatty acid, because the growth-promoting action of summer butterfat and of the fatty acids out of summer butterfat respectively, disappeared upon hydrogenation. This fact cannot be due to a diminished food intake or to a bad resorption of the fat in the ration, having a rather high melting point.

A number of portions of about 2,000 grams of methylesters of fatty acids out of summer butter were submitted to an accurate fractioned distillation. The fraction with 18 C-atoms, which was isolated by means of fractionated distillation of the methylesters of the fatty acids out of summer butter appeared to have growth-promoting properties, the corresponding fatty acid fraction of the hydrogenated fatty acids did not.

Dr. A. J. Kenti, who describes in his recent dissertation at the Amsterdam University the researches leading to the separating of the growth-promoting factor in summer butter, concludes that vaccenic acid seems to possess all the properties mentioned above. Vaccenic acid, discovered in 1928 can be produced from Chinese wood oil.

Prof. E. Brouwer of the School for Agriculture and Forestry of Wageningen, has found that the highest concentration of vaccenic acid is to be found in summer butterfat. This fact explains that the growth-promoting factor of summer butter exceeds the growth-promoting factor of winter butter. A fraction, mainly consisting of vaccenic acid, separated from the fatty acids of summer butter, appeared to bear growth-promoting properties if it was added in a concentration of about 1% to rapeseed oil. Very probably vaccenic acid appears to be one of the growth-promoting factors present in summer butter.

J. Z. BARUCH

Amsterdam-Z.

ABSTRACTS FROM CURRENT LITERATURE

Medicine

Diagnosis of Acute Respiratory Tract Infections. Ziegler, J. E., Curnen, E. C., Mirick, G. S. and Horsfall, F. L.: *Am. J. M. Sc.*, **213:** 3, 1947.

Twenty-six cases of acute respiratory tract infection admitted to hospital during two outbreaks of mild respiratory disease in New York City in the winters of 1943-44 and 1945-46 were subjected to careful clinical and laboratory investigation in an attempt to evaluate the accuracy of the appropriate clinical methods of diagnosis and to enquire particularly into the validity of some of the current conceptions regarding etiology. In addition to the usual history and physical examination the bacterial and virus content of the respiratory secretions were studied together with the bacterial and virus serological reactions.

In the cases described, which appear to be typical of those seen in the numerous waves of respiratory infection which occur each winter in Canada and the United States, a variety of etiologic agents was found. In 7 of the group multiple agents appeared to have been responsible. In 3 of the cases betahæmolytic streptococci were found together with confirmatory serologic evidence of infection. Of 14 cases diagnosed on admission as atypical pneumonia 4 proved to have pneumococcal pneumonia, in spite of low white blood counts and atypical x-ray findings. The sputum in these 4 contained pneumococci of significant types and there were confirmatory serologic findings. Influenza virus was established to have been present in 17 cases although it was suspected in only 8. The suspicion in these 8 proved correct in 7. Primary atypical pneumonia was suspected in 10 cases, the suspicion proving correct in 8 with 6 additional cases in which the disease was an unanticipated finding.

The criteria by which the presence of influenza was correctly suspected was the occurrence of an abrupt onset together with marked early constitutional reaction, and there was known to be influenza virus in the two outbreaks.

In concluding their article the authors discuss the question of the terminology to be used in labelling those cases in which, following clinical and laboratory investigation, it is not possible to come to a definite etiologic diagnosis. They advise the terms "acute respiratory tract infection" and "acute respiratory tract infection with pulmonary involvement". In advocating detailed attention to etiologic diagnosis the authors deprecate the tendency to give antibiotics or sulfonamides as a means of therapeutic testing, pointing out the confusion in diagnosis which this method occasionally causes. Furthermore, they believe that notwithstanding the present tendency towards therapeutic agents of wide efficacy range, future means of treatment may have much narrower fields of application. G. A. COPPING

Insensitivity to Epinephrine in a Patient with a Functioning Tumour of the Adrenal Medulla. Maycock, R. L. and Rose, E.: *Am. J. M. Sc.*, **213:** 3, 1947.

These authors report in detail a case of pheochromocytoma in which it was noted that the patient's reaction to adrenaline was markedly below that seen in normal individuals. Prior to operation for removal of the tumour, injections of quantities of epinephrine up to 1.5 c.c. of 1:1,000 solution failed to cause an elevation of blood pressure or to elicit any other of the usual responses and it was only when 2 c.c. was given that the pressure rose to 162/98. With 2.5 c.c. there was a response to 220/114. After removal of the adrenal neoplasm 0.25 c.c. caused a rise of blood pressure to 172/98 together with the other usual features of adrenaline injection. Further greater amounts caused progressively more marked response.

It has been previously observed that the blood-stream level of adrenaline in patients with pheochromocytomas during the intervals between their attacks is higher than normal. There is, therefore, an insensitivity to adrenaline and it is suggested that this be tested for as a diagnostic procedure. Starting with small amounts increasing injections might be given and the blood pressure and other responses followed. G. A. COPPING

Notes on the Treatment of Tetanus. Graham, J. R. and Scott, T. McN.: *New England J. Med.*, **235:** 846, 1946.

The problem of tetanus is discussed in the light of experience with ten German prisoners stricken with the disease. The great prophylactic value of tetanus toxoid is proved by the rarity of clinical tetanus among the European American army (only one case being reported up until February, 1945) and its comparative frequency among the German wounded whose only prophylaxis consisted of antitoxin at the time of injury.

Proper prophylaxis at time of injury, in persons previously actively immunized with tetanus toxoid, consists of a booster dose of one c.c. In the absence of previous active immunization, three weekly doses of tetanus antitoxin should be administered, as a single dose of antitoxin may be insufficient.

It is important to think of tetanus in all wound cases as the onset may be insidious. Once the disease develops constant care by capable personnel in suitable surroundings is necessary. All aspects of each individual case should receive attention. Large doses of antitoxin (120,000 to 180,000 units) should be administered along with radical debridement of wounds or even amputation. A stomach tube left in place provides an excellent means of supplying the large electrolyte, fluid, caloric and protein requirements, as well as sulfonamides, vitamins, sedatives and mineral oil.

Sedation should be deep enough to make the patient drowsy but should not be carried to an extreme degree because of the danger of pulmonary complications. Paraldehyde and sodium amytal are excellent drugs for this purpose. Routine use of mineral oil prevents fæcal impaction, which is difficult to handle in these patients. NORMAN S. SKINNER

Present Status of the Problem of Amœbiasis. Albright, E. C. and Gordon, E. S.: *Arch. Int. Med.*, **79:** 253, 1947.

Many medical men are unaware that the threat of amœbiasis is a very serious matter. Large numbers of troops are serving in infected areas and others are bringing the infection home. Many cases are unrecognized and untreated because of the insidiousness of the disease or because the intestinal symptoms are common to other conditions. The term amœbiasis includes asymptomatic invasion of the colon by the *Endamœba histolytica* as well as cases with severe symptoms resulting from infection of the bowel wall and liver. Over-all incidence in the United States is estimated at 10%.

The life cycle of the parasite consists of two stages: motile vegetative or trophozoite form and the non-motile cyst. The first invades the bowel wall and other tissues. Encystment is the defensive form in which the parasite continues its existence under unfavourable circumstances. The cyst then is the means of transmitting the infection. Where sanitary conditions are good and water not a menace, food handlers often supply transportation. The mechanism of invasion is the secretion of a cystolytic toxin which dissolves the mucosa of the bowel and enables the parasite to invade the tissues, usually the cæcum first, rectosigmoid areas and later the flexures of the colon. There is little inflammatory reaction and so little scarring if treatment is begun early. Involvement of the appendix may closely imitate acute appendicitis. The asymptomatic case with cysts is the carrier. Although without symptoms the carriers are not devoid of disease so that these strains are not avirulent. It is true however that treatment at this time is most effective.

Diagnostic procedure includes a careful history, proctoscopy, examination of stool, even x-ray study. Any sign of bowel irritation is important in the history. In the examination any fixation of the right diaphragm is important. Tenderness over any part of the bowel is a late finding as is tenderness over the liver and signs of liver enlargement. Proctoscopy shows bleeding points, pus, ulcerations (often pinpoint) sometimes scattered, of whitish colour with a bright red border. Specimens are collected in warm saline and examined before cooling. Hepatitis if diagnosed early will respond to medical treatment. It causes a high white cell count and the patient is almost always free of jaundice. The therapeutic test of treatment with emetine hydrochloride is often conclusive. This drug is most useful in disposing of the *Endamœba histolytica* in the trophozoite stage while several others chiniofon, diodoquin, vioform and carbarsone act on the parasite in the lumen of the bowel.

P. M. MACDONNELL

Relative Clinical and Hæmatologic Effects of Concentrated Liver Extract, Synthetic Folic Acid and Synthetic 5-Methyl Uracil in the Treatment of Macrocytic Anæmias in Relapse. Frommeyer, W. B. and Spies, T. D.: *Am. J. M. Sc.*, **213:** 2, 1947.

Since the first attempts in 1928 to find extracts of whole liver which might be active in the treatment of the macrocytic anæmias, many and varied substances have been investigated. The most potent to date has been concentrated liver extract, and in this paper the authors compare the action of the synthetic products folic acid and 5-methyl uracil with it. The responses of 100 patients to liver extract, 24 to synthetic folic acid and 14 to 5-methyl uracil were studied and observations typical of the whole are given in the detailed presentation of four cases, one of pernicious anæmia, one of nutritional anæmia and two cases of sprue.

In pernicious anæmia the reticulocyte rise and the red cell increase occurred most rapidly and reached the highest level with concentrated liver extract, folic acid and the uracil compound following in that order. Improvement in well-being was earlier with both liver extract and folic acid while glossitis was not bettered and improvement in gastrointestinal symptoms was fleeting with uracil therapy. Folic acid and uracil were almost without effect on the paræsthesias of pernicious anæmia.

In the case of nutritional macrocytic anæmia described as typical of the group the responses of liver extract, folic acid and 5-methyl uracil were similar to that obtained with pernicious anæmia.

In sprue, in which only folic acid and 5-methyl uracil were used, the former was considered to give an earlier improvement in the blood response and to be accompanied by a subjective response which did not follow treatment with uracil compound.

The authors decide in favour of liver extract as the most desirable of the three agents for the treatment of macrocytic anæmia, although consider folic acid to hold an important second place. The failure of folic acid and of 5-methyl uracil to influence the neutral complications of Addisonian anæmia is noted.

G. A. COPPING

Treatment of Shock Accompanying Myocardial Infarction. Schwartz, W. B.: *Am. Heart J.*, **33:** 169, 1947.

The high incidence of "shock or a shocklike state" following coronary thrombosis is generally acknowledged. In one series of 135 cases of coronary thrombosis (Masters *et al.*) manifestations of shock were reported to be present in 50% of the cases. Whether these findings are indicative of a true peripheral vascular collapse or of a sudden development of cardiac insufficiency is a matter of controversy. Some authors state that there is a reflex mechanism initiated by the infarction which leads to the peripheral collapse. Others argue that a decreased blood supply to the tissues initiates the condition. In a series of 59 cases of coronary thrombosis, Fishberg *et al.* noted that the development of shock was usually associated with a decreased return to the heart. There was a decreased circulating blood volume and the venous pressure was lower than normal. These are the findings of peripheral circulatory collapse.

Specific anti-shock therapy in coronary thrombosis is generally not mentioned in the literature or it is discouraged. However, it is noted by a number of authors how poor the prognosis is if the systolic blood pressure during an attack falls to 80 mm. or below. Recoveries take place but they are uncommon. In the case presented as one of acute myocardial infarction in a man of 65, marked manifestations of peripheral collapse occurred, the patient became moribund and the blood pressure was unobtainable; 2,200 c.c. of blood and plasma were administered in 40 minutes and the blood pressure suddenly rose to 140/105, and the intravenous therapy was stopped. Despite the fact that cardiac decompensation had been a prominent feature 4 weeks previous to this episode, no evidence of decompensation occurred following this therapy. In view of the above theoretical considerations and the illustrative results of this case the author advocates that more consideration be given to intravenous therapy in shock accompanying myocardial infarction.

A. L. JOHNSON

Trichinosis: Report of an Epidemic. Hathaway, F. H. and Blaney, L.: *Ann. Int. Med.*, **26:** 250, 1947.

Trichinosis in epidemic proportions was studied in a group of 587 German prisoners of war. Eighty-three were observed in hospital; 437 who were not hospitalized had blood smears taken approximately one week following the last improperly prepared meal in the prison mess. The presence of eosinophilia in a high percentage of the men not hospitalized suggested the presence of trichinosis in a mild or subclinical form in 100 individuals. On the basis of eosinophilia of 10% or greater, it was apparent that 80 men actually were infected regardless of the absence of symptoms.

In this epidemic, which could be considered mild, the typical clinical picture was characterized by a sudden onset of chilliness, fever, malaise, and frontal headache. Associated with these complaints or occurring shortly afterwards there was puffiness of the eyelids which generally lasted three to five days. In most instances muscle pain, or muscle pain with tenderness, occurred a few days later. In the typical case clinical muscle pain and tenderness had disappeared or diminished considerably at the time of discharge. Average hospital stay was 26 days. Gastro-intestinal complaints, mild in degree, occurred as the initial symptom in less than half of the hospitalized cases. The outstanding physical findings were fever and swollen eyelids. Of the 77 patients who complained of muscle pain, associated tenderness was noted in 48.

The most significant laboratory finding was eosinophilia which ranged as high as 61%. There was no constant relationship between the height of eosinophilia and the leukocyte count. However, at some time during hospitalization 69 patients had leucocytosis greater than 10,000.

The small number of muscle biopsies done provided valuable confirmatory information. Skin tests were of value in 68 of 81 patients in which they were performed. No complications of trichinosis were observed in this series and no deaths occurred.

S. R. TOWNSEND

Surgery

Implantation of Fascial Strips Through the Masseter Muscle for Surgical Correction of Facial Paralysis. Owens, N.: *Pl. & Reconst. Surg.*, **2:** 25, 1947.

Two surgical approaches to relief of facial palsy are (1) use of muscle transplants, and (2) support by fascial strips. The author reports 11 cases treated by looping fascial strips through the masseter muscle. Three strips are inserted: (1) from beyond the philtrum on the sound side to the masseter on the paralyzed side; (2) from beyond the mid line of the lower lip on the sound side to the masseter on the

paralyzed side, and (3) from the angle of the mouth on the paralyzed side to the masseter on the same side. These strips are drawn tight enough to pull the lip and angle of the mouth upward about 1½ cm. and backward approximately 2½ cm. The lip is supported by gauze and collodion dressing for 16 to 21 days.

Exercises involving the masseter muscle are then started. The use of a mirror while exercising aids in developing co-ordination. All 11 cases, following complete healing, reflected improvement from the standpoint of support and rather satisfactory improvement as regards re-animation. STUART GORDON

Postoperative Pulmonary Atelectasis. Kruger, A. L., Marcus, P. S. and Hoerner, M. T.: *Am. J. Surg.*, **73:** 531, 1947.

Atelectasis is a serious postoperative complication in itself and its complications, pneumonia and bronchiectasis, increase its importance. It may follow any operation and any anæsthetic and its incidence was 0.45% in a series of 6,553 major surgical procedures.

Impaired cough, with retained bronchial secretions causing mechanical bronchial obstruction is the favoured theory of etiology. Sedatives, especially morphine, may be used too freely. Operations lasting over an hour are more frequently followed by atelectasis. The symptoms are cough, shortness of breath, chest pain, blood-streaked sputum and cyanosis, in order of decreasing frequency. Fever between 101 and 104° F., pulse over 100 per minute, respirations over twenty per minute accompany signs of diminished chest expansion, fremitus, breath sounds and râles. X-ray plates show lobular, lobar or massive haziness and in lobular atelectasis, tracheal shift and elevation of the diaphragm is not seen. Suspicion of the existence of atelectasis is the most important factor in its early diagnosis.

The prevention of atelectasis by postoperative frequent turning, deep breathing exercises, CO_2 inhalations, encouragement of coughing is important and it is less if elective procedures are not done in patients with upper respiratory infections or asthma. Once the diagnosis is made, immediate efforts must be made to remove the obstructing agent so that the affected lung section is completely reaerated. If simple efforts fail, bronchoscopic suction usually effects dramatic results. A carbon dioxide and ether technique is described as being valuable. BURNS PLEWES

Studies on the Acid Debridement of Burns. Sulyberger, M. B., Kanoff, A. and Baer, R. L.: *Ann. Surg.*, **125:** 418, 1947.

Early removal of slough has a beneficial effect in third-degree burns and other necrotic wounds. Such removal should be as "non-shocking" as possible, and must not harm viable tissue. In 1944 Connor and Harvey reported the use of pyruvic acid in a water-starch gel for removal of slough. The authors undertook studies to (1) compare the effect of this method with the effect of other methods in the topical therapy of standard third-degree burns in human subjects; (2) elaborate a more practical vehicle; (3) find a more stable and generally available acid and (4) to extend the sphere of knowledge regarding the technique.

Several hundred young male volunteers at a disciplinary barracks were used. Two symmetrically situated areas, one on the flexor surface of each forearm, were burned. Early experiments were done using liquid mustard gas to produce the burns; later with heat.

The first series of experiments confirmed the findings of Connor and Harvey. The superiority of the acid debridement method was maintained through the entire series of over 1,500 wounds. Later experiments indicated that the effects of acid debridement are not specific for pyruvic acid, but appears to be dependent on the maintenance of the correct supply of hydrogen ions over a sufficient period of time. 0.1M phosphoric acid proved to be the best among other acids tested.

It was found possible to replace the starch paste vehicle. Several formulæ are given in the communication. Final studies demonstrated that certain forms of dry hygroscopic powders containing the acids could be prepared. Suitable gels formed on the addition of the proper amount of water. STUART GORDON

Treatment of Deep Venous Thrombosis with Reference to Subcutaneous Injection of Heparin and Use of Dicumarol. Holden, W. D.: *Arch. Surg.*, **54:** 183, 1947.

The enthusiasm of various clinics for the practical use of venous ligation, anæsthesia of the paravertebral lumbar sympathetic system and the anti-coagulants has awakened interest in the therapy of deep venous thrombosis but a considerable ambiguity in the minds of many physicians as to the benefit from the use of any one of them. The object in treating deep venous thrombosis of the lower extremities and pelvis is to prevent the occurrence of pulmonary emboli and to minimize the late effects of venous obstruction.

Both arteriospasm and venospasm associated with venous thrombosis can be abolished by paravertebral lumbar block. Ligation of the superficial and common femoral veins, the iliac veins and the vena cava has proved to be of immense value in reducing the incidence of pulmonary infarction. The indications for ligating the superficial femoral veins are clear and those for the more proximal veins ambiguous. If caval or iliac ligations are to be performed, they should be done by surgeons experienced in vascular surgery, as Homans has advised. The intravenous administration of heparin has been difficult because of the necessity of frequent determinations of the clotting time, the repeated intravenous injections and the maintenance of a constant intravenous infusion. The heparin-Pitkin menstruum, given subcutaneously, offers a more practical method of administration. Clinical evidence has shown that the incidence of pulmonary infarction is reduced by either the prophylactic or the therapeutic administration of heparin. Dicumarol has been shown to be effective in preventing the occurrence of postoperative venous thrombosis. The most effective level of the prothrombin clotting time is not known. The author believes that beneficial effects, *i.e.*, cessation of the propagation of a thrombus, are realized when the prothrombin clotting time is between 30 and 50% of normal. At this level, however, there is no prolongation of the actual clotting time. G. E. LEARMONTH

Obstetrics and Gynæcology

Timing of Endometrial Biopsy. Hadley-Jackson, M.: *J. Obst. & Gyn. Brit. Emp.*, **54:** 86, 1947.

Thirty-seven biopsies are included in the series; 7 of these were done before the 14th day, that is in the first half of the cycle and most probably before ovulation had occurred; 30 were taken on or after the 14th day of the cycle and 18 of these between the 25th and 35th day, therefore probably after embedding had started. The microscopical appearance corresponded on the whole with what would be expected for the days in the cycle when the biopsies were taken, except in a few cases where the stage of development was either rather more or less advanced than might have been expected. For example, the biopsy taken on the 11th day of the cycle in one case shows some subnuclear vacuolation, and in those taken on 23rd, 24th and 25th days the glands are markedly tortuous, secretion is plentiful and there is some predecidual change in the stroma; in fact these 3 sections are comparable to some of those taken more than a week later. On the other hand, the biopsies taken on the 14th and 25th days in a case are apparently at least a week behind in their development. It may be noted that the greatest variations in degree of development is found in those biopsies taken between the 21st and 26th days of the cycle. P. J. KEARNS

Preliminary Report on the Pelvic Brim Index. Heyns, O. S.: *J. Obst. & Gyn. Brit. Emp.*, **54**: 39, 1947.

The author gives an extensive and unique discussion on pelvic growth, shape, and conformities, with etiological factors concerned. He asks the question—What are the anatomical factors that influence the brim index? These factors appear to be as follows: (1) Growth of bone at certain epiphyses: (*a*) Acetabular epiphyses, but more directly the anterior limb of the Y-shaped epiphyses. (*b*) Epiphyses at the symphysis pubis. (*c*) Sacral epiphyses concerned with growth in width in the region of the sacroiliac point. (2) Development of the sacral alæ causing greater anterior curvature of the sacrum, and thus a pushing of the sacrum posteriorly or the rest of the girdle anteriorly. (3) (*a*) Bending, possibly due to rickets, of the bony girdle at the iliac portion of the ilio-pectineal line. This will increase the length of the greatest transverse diameter of the brim. (*b*) Wherever the bending of bone mentioned in (3*a*) occurs as the result of an antero-posterior force, the conjugate diameter will be diminished. (4) The tendency of the sacral promontory to be situated forward, *i.e.*, near to the greatest transverse diameter, in pelves of the android type. P. J. KEARNS

Radiology Use in Predicting Difficult Labour. Moir, J. C.: *J. Obst. & Gyn. Brit. Emp.*, **54**: 20, 1947.

Obstetrical radiology has been criticized because it emphasizes certain features of the birth process and ignores others that also play a highly important part in deciding the course of labour. This, of course, is perfectly true, and we must always be on guard against investing radiological reports with unreasonable importance. Nevertheless, disproportion in its widest sense is chief among the causes of difficult childbirth, and here it is that radiology gives most help. If we trouble to take pelvic measurements at all we show our belief in their value. And, if we so believe, then surely it is right to strive for fuller knowledge and greater accuracy. The fear is sometimes expressed that increased use of radiology will lead to increased and unnecessary surgical interference. This is possible when bad radiology is combined with bad obstetrics. But a similar fear might equally well be expressed when radiology is used as an aid to the diagnosis and treatment of fractured bones, of alimentary disorders, of pulmonary disease and so on. Yet radiology is accepted without question as a useful and even indispensable help in these conditions. It is further said that an increased use of radiology will lead to a weakening clinical judgment and that the radiologist will displace the obstetrician in deciding the manner in which delivery shall be conducted. This criticism again supposes that the obstetrician has no ability or clinical judgment of his own, and again in reply it may be said that if it is true that radiology has a stultifying influence in obstetrics, then it must have a like influence in other branches of medicine, as, for example, in the diagnosis and management of cases of pulmonary tuberculosis. In point of fact the fear is groundless. The obstetrician's clinical judgment is bettered, not worsened, by his increased knowledge and his clearer mental picture of the happenings in the patient's pelvis. P. J. KEARNS

Technique of the Lower Segment Cæsarean Section. Uhma, C.: *J. Obst. & Gyn. Brit. Emp.*, **54**: 65, 1947.

The author emphasizes some facts of particular importance. They concern first the detachment of the bladder. The incision of the vesico-uterine fold of the peritoneum should not be too close to the peritoneum where it is firmly attached to the uterine wall. This makes it much more difficult to find the right layer between the bladder and uterine wall and often causes the cutting of blood vessels. Later this hinders the operation. If the incision in the peritoneum is performed about ½ to ¾ of an inch away from the firmly attached peritoneum then it exposes the very loose connective tissue which does not contain large blood vessels. The latter extends between the dissecting forceps which draws up the bladder and the uterine wall and is cut half way with scissors in the direction of the uterus. This leads to the right layer and later permits of the bladder being detached from the lower segment by blunt dissection. The bloodless exposure of the lower segment prevents the formation of small hæmatomata in the connective tissue, gives a clear field for the operation and contributes to clean healing. The next important point to be considered is the making of a longitudinal fold in the lower segment wall. This fold is held in position by clamping it with two Allis's forceps and making a small incision between them. The blood is thus prevented from flooding the incised surface and the liquor amnii is drawn off through this small opening and is almost entirely prevented from entering the peritoneal cavity. This is of importance if the liquor amnii is infected. Finally it is of great importance to wait until the injection of pituitrin given into the active part of the uterus has contracted the uterine muscles well before removing the placenta. P. J. KEARNS

Fibrous Nature of the Human Cervix, and its Relation to the Isthmic Segment in Gravid and Non-gravid Uteri. Danforth, D. N.: *Am. J. Obst. & Gyn.*, **53**: 541, 1947.

In the non-pregnant human uterus the cervix is found to be composed predominantly of fibrous connective tissue with an average of only about 15% smooth muscle. The tissue superior to the cervix is composed predominantly of smooth muscle. The transition from fibrous to muscular tissue is generally quite abrupt; but it may be gradual, occurring over the space of 5 to 10 mm. The non-pregnant isthmus is an indefinite, variable segment which is composed principally of smooth muscle, which is lined by a transitional epithelium, which is bounded below by the fibrous cervix and which above blends imperceptibly with the remainder of the uterine musculature.

In the pregnant uterus the length of the cervix does not change significantly during early pregnancy. In the third month of pregnancy the isthmus elongates. This is considered as a hypertrophic response to pregnancy. When the elongation occurs the products of conception are confined to the portion of the uterus which is superior to the isthmic canal. When they have enlarged to require more space, the isthmic musculature unfolds to accommodate them and thus comes to make up a part of the wall of the ovum chamber. The unfolding is limited inferiorly by the fibrous cervix.

It is concluded that from both anatomic and functional standpoints the isthmus forms a unit with the remainder of the uterine musculature. It is believed that the concept of the isthmus uteri as a separate, distinct entity should be eliminated and that, rather, the uterus should be considered as being composed of two major parts, corpus and cervix, according to whether the fundamental structure is chiefly muscular or chiefly fibrous. The isthmus is considered as part of the corpus, specifically, the part of the corpus which lies between (*a*) the level of the fibromuscular junction of the cervix with corpus and (*b*) the plane of the inferior level of the uterine cavity. ROSS MITCHELL

Irregular Shedding of the Endometrium. McKelvey, J. L. and Samuels, L. T.: *Am. J. Obst. & Gyn.*, **53**: 627, 1947.

Irregular shedding of the endometrium is a specific form of functional uterine bleeding. It is an abnormality of true menstruation. It is characterized clinically by prolonged and profuse menstrual bleeding and histologically by retention over variable periods of time of abnormal amounts of endometrium which has functioned. The endometrium shows abnormalities of the basic endometrial features of menstruation in various degrees of retardation of shrinking, shedding, involution of glands and stroma and of healing. Sodium pregnandiol glucuronidate is excreted in the urine during the

time of the uterine bleeding. This is not seen in other conditions, and seems to be characteristic of irregular shedding of the endometrium. Histologic diagnosis is essential, and requires timing of the curettage in relation to the expected duration of the bleeding. Clinical experiences with 34 cases of irregular shedding are briefly summarized. ROSS MITCHELL

Management of Delivery Following Stillbirth from Previous Dystocia. Hunt, A. B. and DeVoe, R. W.: *Am. J. Obst. & Gyn.*, **53:** 812, 1947.

The deliveries of 32 patients subsequent to stillbirth from dystocia in primary pregnancies are reviewed. These patients were seen at the clinic from January 1, 1936 to July 1, 1946. A gross fetal mortality of one fetal death in 36 deliveries or 2.8%, was obtained. This infant was dead in the uterus because of severe toxæmia of the mother on her admission to the hospital. There was no maternal mortality in this series. When these data are added to those reported ten years ago it is found that 64 women had lost 71 babies from dystocia before this study was made. Subsequently they were delivered of 86 babies with three fetal deaths, a gross fetal mortality rate of 3.5%. There was one maternal death early in the first series. The maternal mortality rate for subsequent deliveries was therefore slightly more than 1%. Only two of the 64 women including the one that died are without normal living infants. One of these had normal delivery but lost her infant some months later from nonobstetric causes. She is now pregnant and the chance for a living baby is excellent.

The opportunity for *individual* prenatal care, examination and study of the safest method of delivery is most valuable in the care of these patients. In these cases the conduct of the first delivery subsequent to the stillbirth from dystocia seemed to determine the outcome in later deliveries. Patients who were delivered successfully through the pelvis continued to have this type of delivery and those who had to submit to abdominal delivery again required this management. ROSS MITCHELL

Meningitis Following Continuous Caudal Anæsthesia. Brown, W. W.: *Am. J. Obst. & Gyn.*, **53:** 682, 1947.

Clinical signs and symptoms of acute meningitis developed twenty-four hours after cessation of caudal anæsthesia continued for nearly six hours in a previously normal gravida iii in premature labour. No evidence of infectious focus could be demonstrated elsewhere in the body. The spinal fluid of three selected normal patients, each carried for more than six hours on continuous caudal anæsthesia was grossly and microscopically negative. This suggests that neither metycaine nor the plastic catheter was irritating to the meninges of these three patients. This is one of the few, and perhaps the second, case of meningitis reported following continuous caudal anæsthesia. ROSS MITCHELL

Penicillin and Acute Puerperal Mastitis. Hodgkinson, C. P.: *Am. J. Obst. & Gyn.*, **53:** 834, 1947.

Penicillin, if given sufficiently early, will prevent suppuration in acute staphylococcic puerperal mastitis. Because penicillin is not secreted in the milk in sufficient amounts to control contaminating staphylococci, it is felt advisable to inhibit lactation to prevent reinfection of the breast. Sulfonamide therapy is not recommended as a substitute for early penicillin therapy. ROSS MITCHELL

Industrial Medicine

Clinical Experience with Chemical Hazards in Industry. Gehrmann, G. H.: *N.Y. State J. Med.*, **46:** 2409, 1946.

That proper methods of control will result in safety in handling of materials, no matter how toxic they are, is stressed by the author of this article. He maintains that where dangerous chemical compounds are found in use, it is usually ridiculous to recommend a substitution of a non-toxic material. The answer to the problem lies in devising ways and means of manufacturing and medically controlling the workers in the toxic operation. Every problem of exposure hazard must be approached with careful and systematic means of clinical observation.

In any well-organized industry, preventive medicine should be the program of primary importance. Industry needs more and better diagnostic methods that will indicate early physiologic changes, not clinical methods to determine pathologic damage.

In discussing the general problems encountered in connection with industrial chemical hazards, the author presents information which he has gained entirely by experience, and which he feels will be helpful in planning preventive medical procedures. He refers to certain procedures in industry which have been accepted as suitable, and shows the pitfalls and serious difficulties which can be encountered. Among the procedures discussed are (1) the determination of so-called safe allowable concentrations of dusts and gases; (2) the installation of ventilation for the removal of dusts and gases; and (3) the introduction of various types of protective creams as a method for control of industrial dermatitis. In connection with the latter he stresses that before the introduction of any protective cream into any operation, it is essential that one have a thorough understanding of the toxicity of the material at hand and accurate knowledge as to whether it can be absorbed through the skin. If it can, other methods of protection must be devised. MARGARET H. WILTON

The Physically Impaired Worker in Industry. Kossoris, M. D. and Hammond, H. S.: *Monthly Labour Rev.*, **63:** 918, 1946.

Although the successful utilization of physically impaired workers in industrial occupations has received much attention throughout the country, a large segment of industry still discriminates against them. Impaired workers do not want charity; they want places as useful and respected members of the community. Their success depends on whether industry will accept the policy of hiring them for what they can do well in place of rejecting them because of what they cannot do.

In order to determine to what extent such discrimination is justified the Bureau of Labour Statistics, at the request of the Veterans' Administration, undertook to appraise the work performances of about 10,000 seriously impaired workers. Ten specific types of physical impairments were defined by a committee of industrial physicians; these were ones regarded to be so severe that they would raise serious difficulties for persons seeking employment. Industry's own records supplied information regarding work performance, absenteeism and injury experience. Each impaired worker selected for appraisal was matched against two, sometimes three, unimpaired workers of about the same age, sex and work experience, on the same shift and performing the same job in the same department of the plant.

It was found that as a group the impaired were 2% more productive than the unimpaired. An analysis of detailed data showed that 70% of the impaired were as good or better than the unimpaired in the corresponding control group. The absenteeism rates of the two groups were identical; each lost 3.8% of scheduled working hours. Among the types of absenteeism there were no significant differences.

The disabling accident record of the so-called "normal group" was about 40% worse than that of the impaired group, doing exactly the same work, and exposed to the identical work hazards. The authors believe that this is due to the fact that the impaired workers are careful not to get hurt again. Furthermore there was very little difference in average time lost per injury between the two groups. MARGARET H. WILTON

Dermatology

Leukæmia Cutis. Report of a Case with a Discussion of Treatment by the Use of Hormones. Robey W. H. and Gardiner, R. G.: *New England J. M. Sc.*, **236:** 505, 1947.

The case described is one of leukæmia cutis in a 51-year old married man, 2½ years elapsing from appearance of symptoms until death. Insidious onset with slight punctate erythematous rash, with mild itching, on the trunk, progressed in 3 months to a generalized appearance of mild sunburn. Swelling of superficial lymph-nodes which appeared about this time increased rapidly. Histological examination of these supported a diagnosis of lymphatic leukæmia although with the exception of a relative increase in lymphocytes, a large proportion of which were young and atypical forms, the blood-count was normal. The condition continued to become worse with large doses of navitol. Both regional and general x-ray treatment with continued navitol failed to delay the general deterioration, and in the 10th month of the disease all other treatment was replaced by administration of progynon B. Improvement, notably a decrease in the general anasarca, was rapid. Two months later diethylstilbœstrol was added. A month later the patient resumed a limited amount of business activity and played 18 holes of golf with ease. Three months later, such was the improvement, that progynon was stopped. A few weeks later a fine petechial rash appeared on the upper trunk, and although there was again some improvement on resumption of progynon, his condition 5 months later was much worse than when the progynon had been stopped. Death occurred suddenly 3 months later and autopsy showed leukæmic infiltration of spleen, liver, kidney, bone and skin with thrombus of an iliac vein, pulmonary embolism and lung infarct. The breasts showed rather marked hypertrophy.

It is admitted that the case might have improved under some other form of therapy or none, but the symptoms diminished only when hormones were given, and it is recommended that in similar cases this form of therapy be given serious consideration.

D. E. H. CLEVELAND

Recent Advances in Cancer Therapy. Haagensen, C. D.: *Bull. N.Y. Acad. Med.*, **23:** 123, 1947.

The author states that it is now clear that with the exception of a few types of cancer, such as some forms of epithelioma of the skin, epithelioma of the larynx and epithelioma of the cervix, radiation cannot compete with surgery as a curative agent. The last-mentioned he considers the one disease in which radiation has become the preferred method of treatment. He believes that cancer of the skin is better treated in the initial attack by surgery for the following reasons: surgery incapacitates the patient for a shorter time; it gives a better cosmetic result and is more certain to cure. The radiotherapist is especially at a disadvantage where the lesion overlies bone or cartilage, in which structures radiotherapy is unsuccessful. D. E. H. CLEVELAND

Skin Tumours. Cannon, A. B.: *Bull. N.Y. Acad. Med.*, **23:** 163, 1947.

There are three main types of skin cancer: The basal cell epithelioma which is non-metastasizing, the squamous cell epithelioma which metastasizes, and the malignant mole. While the different basal cell types may differ in appearance and local destructiveness, the outstanding feature which they have in common is the pearly or waxy telangiectatic margin or rim. The "rodent ulcer" is the most destructive form, although it may take several years to do much damage. It must be distinguished from syphilitic gumma, tuberculosis, blastomycosis and lupus, and while it is usually identified by its waxy or pearly nodular border and red, bleeding, granular base, a biopsy makes the diagnosis conclusive. The author mentions having seen cases of gumma recently treated by radiation for carcinoma with severe and sloughing reactions. (*The reviewer has encountered similar cases!*) The multiple benign superficial epitheliomas of covered parts often occur in patients who have been under treatment with arsenic in earlier years (for non-cancerous, usually cutaneous conditions). In many instances arsenic in excess may be demonstrated in the blood and urine.

Squamous cell carcinoma, while relatively benign and usually accessible may be neglected or mistreated resulting in spread by metastasis and terminate fatally. It appears more often on the extremities and exposed parts, especially at the site of chronic irritation, such as radiodermatitis, scars, chronic ulcers and arsenical keratosis. Not all carcinomas occurring in x-ray burns are squamous cell types, both basal cell and basosquamous being seen. The comparatively rapid growth, inflammatory reaction, irregular sloughing and ulcerating base and hard border are characteristic. All mucous membrane epitheliomas are of the squamous cell variety. A biopsy before treating epithelioma is imperative.

Prophylactic measures include the early recognition of conditions from which epithelioma may develop. The majority of epitheliomas are found in persons over 40 and they occur more often in men than in women. Chronic seborrhœal redness and scaling in the nasolabial folds often is followed by epithelioma. The aging skin should be protected by thorough cleansing with water and a bland soap, followed by the use of a suitable cream, a protective powder and avoidance of over-exposure to sun and wind. D. E. H. CLEVELAND

OBITUARIES

As we go to press we learn with deepest regret of the death in England of Dr. W. B. Howell. Fuller notice will appear next month.

Dr. Harry Bertram Anderson, aged 78, died on May 21, in Toronto. He practised medicine in Toronto for 40 years. He had served on the staffs of Toronto General and Grace Hospitals, Muskoka Sanatorium, Queen Elizabeth Sanatorium and the Hospital for Sick Children. He also held professorships in pathology and clinical medicine at both Trinity College and the University of Toronto.

A native of Peterborough County, Dr. Anderson was a gold medallist graduate of Trinity College in 1892. He took postgraduate study in London, Eng., Baltimore, Munich and Vienna and was a charter member of the F.R.C.P. and also a L.R.C.P. and M.R.C.P., London, Eng.

During the First Great War he served as a lieutenant-colonel with the R.C.M.C., organized the staff of Central Military Hospital, Toronto, and acted as consultant for the Spadina Convalescent Hospital. Dr. Anderson was a past president of the Toronto Pathological and Clinical Societies, the Academy of Medicine, the Ontario Medical Association, the Æsculapian Society and the University of Toronto Medical Alumni Association.

He was a member of Lambton Golf Club, Toronto Hunt Club, Ionic Lodge, A.F. and A.M., St. Paul's Chapter, R.A.M., and an elder of Old St. Andrew's United Church. Surviving, besides his widow, are three grandchildren; a brother, and a sister.

Major Karl Kenneth Blackadar died in Stadacona Naval Hospital, Halifax, May 7, after an illness of two weeks.

Born in 1890 in Halifax County he attended Dalhousie Medical School, graduating in 1916. On graduation he joined the Dalhousie Medical Unit as a medical officer and spent two years overseas. After his return to Canada

he established private practice at Mahone Bay and later at Meteghan.

In 1927 Major Blackadar joined the Canadian National Steamships as a surgeon and remained with them until shortly after the outbreak of World War II, joining the R.C.A.M.C. in 1942. He was stationed at Yarmouth, Aldershot and Halifax and was known to thousands of army recruits taking their basic training at these centres.

Surviving him besides his widow and his mother, are three daughters and one brother.

Le Dr Maurice Bourgeois, 49 ans, coroner pour le comté de Champlain, est décédé subitement au Cap-de-la-Madeleine, le 12 mai, succombat à une crise cardiaque.

Originaire de la région de Nicolet, il avait fait ses études classiques au séminaire de Nicolet et ses études médicales à l'université Laval, à Québec. Il avait d'abord pratiqué sa profession à S.-Sylvère, sur la rive sud, et était venu s'établir au Cap-de-la-Madeleine il y a une douzaine d'années.

Il laisse son épouse, sa mère, et deux filles.

Le Dr J.-R. Brais, de S.-Jérôme, autrefois coroner pour le district de Terrebonne, est décédé, le 7 mai à l'âge de 58 ans et 9 mois. Outre ses anciennes fonctions de coroner, il avait été président de l'association médicale de S.-Jérôme, président honoraire de l'association des vétérans du comté de Terrebonne, président du comté, etc. Il avait combattu outre-mer, lors de la première grande guerre, comme capitaine.

Il laisse son épouse, deux filles et quatre garçons.

Le Dr J.-B. Drouin, autrefois de Victoriaville est décédé, à Outremont le 18 mai après une longue maladie.

Ex-interne de l'hôpital Notre-dame de Montréal, le Dr Drouin a exercé pendant 38 ans sa profession à Victoriaville où il fut maire durant 8 ans, et il occupa également la charge de coroner du district d'Arthabaska.

Il laisse sa femme, et six filles.

Dr. Thomas Stone Farncomb died on May 17 in Trenton, Ont. Born in 1869, he graduated in medicine from Trinity in 1891.

Dr. Ernest S. Harding, died on June 6 in Montreal in his 70th year. He was for many years the chief medical officer and a member of the board of directors of the Royal Edward Institute.

Dr. Harding was born in Amherst, N.S., and received his education at Acadia University, Dalhousie University and later took his medical degree at McGill University.

He immediately became actively involved in the combat against tuberculosis and formed the Montreal League for the Prevention of Tuberculosis, of which he became head physician. In 1909, the Royal Edward Institute was formed under the patronage of His Majesty, Edward VII. The institute was first located on the present site of the Canadian National Railways Terminus. The first open air school for tubercular children was opened at this location. It was in the form of a day school, and beds were placed on the roof of the building, where the children obtained the sunshine and fresh air. Dr. Harding also instituted the camp for tubercular children at Ste. Agathe, which is still open.

Dr. Hibbert Winslow Hill died on May 23 in Hackensack, Minn. He graduated in medicine from the University of Toronto in 1893.

Dr. Arthur John Lomas, aged 67, superintendent of the University of Maryland Hospital for 16 years until his retirement in 1939, and a graduate in medicine of McGill University, died on May 18.

Born in Montreal, Dr. Lomas was the son of the late Henry Stephen and Mary Lomas who came to Canada from England. Dr. Lomas obtained his medical degree at McGill in 1902, and later obtained a degree in public health from the same university.

After practising in Britain, Africa and as a ship's doctor, he joined the Canadian Army in 1915, and was a member of the staff of a military hospital in Egypt during the Great War. He also served in England and later in the field with No. 3 Canadian General Hospital. His companions will recall his liveliness and good humour. He served in the late war as a major.

Dr. Lomas, who married Elene Marie Perez, daughter of the then Spanish Consul General in Newfoundland, was regarded as "a wizard" in drawing up hospital construction plans. He is survived by his widow.

Dr. William B. MacDiarmid, aged 73, Liberal member of the Commons for Glengarry from 1940 to 1945, died suddenly May 13 of a heart seizure at his home in Maxville, Ont., where he had practised for many years.

Dr. MacDiarmid, was returned in the general election of June 11, 1945, in which Prime Minister Mackenzie King suffered a close defeat in the Saskatchewan riding of Prince Albert. Shortly after Dr. MacDiarmid resigned his seat and accepted a position as surgeon for the R.C.M.P., thus allowing the Prime Minister to run in the resultant by-election and gain a seat in parliament.

A native of Athol, in Glengarry County, he graduated from McGill University in 1900, and for many years was health officer for Roxborough township and Maxville. Surviving are his widow and a son.

Le Dr Ernest MacKay, de Québec, est mort le 5 mai après une longue maladie, à l'âge de 89 ans et 2 mois. Né à S.-Eustache, comté de Deux-Montagnes, il habitait notre ville depuis de nombreuses années.

Il laisse sept fils et une fille.

Dr Lorenzo Martel est décédé le 25 avril, à l'âge de 40 ans.

Il avait poursuivit ses études médicales à l'université Laval et en France. Pendant cinq ans, il servait dans l'Armée active canadienne, à titre de médecin-major du Corps médical, il demeurait encore officier de la réserve du Corps médical. Il était médecin du département des mères nécessiteuses, au ministère provincial de la Santé. Le Dr Martel était membre de l'Association ambulancière St-Jean et du Club de Réforme.

Il laisse pour pleurer sa perte, outre son épouse, trois fils, et une fille.

Dr. Kenneth Inches Murray, aged 54, died on May 17 at Lethbridge following an illness of three weeks. He had been a patient in Galt Hospital following a heart seizure.

He was born in St. Stephen, New Brunswick, where he received his early education. He interrupted his university career to enlist at the outbreak of the First Great War and served in France with the Royal Canadian Engineers for three years. On demobilization he resumed his course at Queen's University in Kingston, Ont., graduating with a medical degree.

In 1919 Dr. Murray first came west, interning in Calgary General Hospital.

He practised at Raymond, Blackie and Coalhurst before starting his practice in Lethbridge in 1935. At the time of his death he was Lethbridge's medical health officer and medical superintendent of Galt Hospital. He was a member of the Lethbridge branch of the Canadian Legion, of the Chinook Club and of the Lethbridge Kiwanis Club. He served on the executive of Kiwanis for some time. He was medical officer for the 18th Field Regt., R.C.A., in Lethbridge and coroner for Lethbridge and district for a number of years.

Surviving are his widow, two brothers and two sisters.

Dr. B. Choné Oliver, who served on medical missions for 45 years in India, died on May 21 in Fort William. Appointed secretary of the Christian Medical Association of India, Burma and Ceylon in 1933, she retired

in 1945. For a time, Dr. Oliver travelled throughout Canada and the United States in the interest of Christian medical work in India. A few months after retirement she was called back to her former post to meet an emergency, but returned to Canada late in 1946 because of ill-health. Dr. Oliver was a graduate of University of Toronto in 1900. For 25 years she served as a medical missionary in hospitals under the W.M.S. in the Central India Mission of the Presbyterian Church in Canada and later United Church. In 1929 she was appointed a secretary of the National Christian Council of India with special responsibilities in the medical field. During her 15 years in office as secretary of the Christian Medical Association of India, Burma and Ceylon, she rendered outstanding service to India and the British Empire in consolidating and strengthening Christian medical work. She was credited with the achievement which raised the standards of training and equipment for doctors and nurses and the development of the Women's Medical College at Vellore, South India, into the Vellore Christian Medical College for men and women. L.A.C.

Dr. Rose Pringle, aged 81, former head of women's department of the Westchester division of the New York Hospital in White Plains, N.Y., died in Detroit May 9.

Dr. Pringle, born in Fergus, obtained her medical degree from Dalhousie University, Halifax, and joined the Westchester division in 1895.

Dr. John R. Stewart, past president of the College of Physicians and Surgeons of Ontario, died at his home in Toronto on May 25, following a lengthy illness. In his 68th year, he had practised medicine for about 40 years.

Born at Waba, in 1879, Dr. Stewart attended Renfrew high school and Queen's University, graduating from the latter with degrees in arts and medicine in 1906. For 13 years he conducted a practice in Cobden, and in 1922 moved to Toronto.

Elected president of the College of Physicians and Surgeons of Ontario in 1945, he took an active part in the Academy of Medicine, and was appointed to a number of committees. He was also a past president of the Toronto East Medical Association. He served as an elder in Old St. Andrew's United Church for many years, and was a member of the Masonic Order and the COF.

He is survived by his widow, one daughter, two sons, one sister and four brothers.

Dr. L. A. S. Stewart, aged 58, well-known Ottawa surgeon and a member of the staff of Ottawa Civic Hospital since 1924, died on May 5. He was born at Aylmer, Que., and took his training at McGill University.

Dr. Edward Roy Tyrer, aged 63, died suddenly at his home in Barrie, Ont., on May 28. He is survived by his widow.

Dr. James Roy West, of Ottawa, died in hospital in Montreal, on May 22, after an operation.

NEWS ITEMS

Alberta

The offices of the Registrar of the College of Physicians and Surgeons and the secretary of the Canadian Medical Association, Alberta Division, have moved from No. 10 Merrick Building to 207 Alexandra Block, Edmonton. This move was necessary in order to obtain more space required for administration of the pensioners' medical service.

The Council and College of Physicians and Surgeons of Alberta, on behalf of the profession, entered into an agreement with the Provincial Government wherein the members of the College are to provide full medical, surgical and obstetrical care to old age pensioners, blind pensioners, recipients of mothers' allowance, and the dependents of each. The Government is to pay the College of Physicians and Surgeons a sum of money, out of which, each service will be paid.

The Alberta Dental Association has entered into a similar agreement with the Government, and has opened its office with the College of Physicians and Surgeons.

The President-Elect, Canadian Medical Association, Alberta Division, Dr. A. E. Wilson, has just completed a tour of the medical districts. Dr. Wilson was accompanied by Dr. H. N. Jennings, of Calgary, Dr. A. J. Fisher, of Calgary, and Dr. W. Bramley-Moore, Secretary of the Division. Dr. Wilson and Dr. Bramley-Moore discussed economic matters especially in connection with the provision of medical care to old age pensioners, blind pensioners, and recipients of mothers' allowance, also the question of introducing a voluntary prepaid medical plan into the Province.

A draft plan on medical care is being prepared and will be presented to the profession at the annual meeting in September.

Dr. H. N. Jennings presented papers on cardiovascular diseases, and Dr. Fisher gave illustrated talks on the value of x-ray examination in obstetrics.

Meetings were held in Medicine Hat, Lethbridge, Drumheller, Red Deer, Camrose, Vegreville and Dunvegan. All meetings were well attended.

The Alberta Government's new hospital and medical services' division will direct the three hospitalization services being granted to old age and blind pensioners and recipients of mothers' allowances and their dependents. The scheme which will cost the Province about $500,000 annually, was approved at the last session of the legislature.

Construction of a $300,000 hospital extension will soon be in progress at Camrose. The hospital is operated by the Sisters of Providence. This extension will increase the hospital's capacity to more than one hundred beds and will be near the former Camrose Normal School, which the Provincial Government is converting into an old peoples' home.

G. E. LEARMONTH

British Columbia

The Vancouver Medical Association held its Annual Summer School from June 2 to 6 inclusive. Lectures were given at the Hotel Vancouver, clinics at the hospitals.

The program this year was an excellent one, and fully maintained the high standards of this School. The speakers were: Dr. Alfred J. Elliott, Professor of Ophthalmology, University of Toronto; Dr. D. Nelson Henderson, Department of Obstetrics and Gynæcology, University of Toronto; Dr. Arnold S. Jackson, Jackson Clinic, Madison, Wis., U.S.A., Lecturer in Surgery; Dr. Clement A. Smith, Professor of Pædiatrics, Harvard Medical School; Dr. Cyrus C. Sturgis, Professor of Medicine, University of Michigan.

The Hamilton report on hospital matters in Vancouver has recently been received. It has not yet been fully made public, but certain features of it have appeared in the press. It recommends, among other things, that the Vancouver General Hospital be greatly enlarged, and that a part of its bed capacity be made available for clinical teaching, to the medical school whose formation is contemplated by the University of British Columbia. It seems likely, from remarks made by the Ministers of Education for B.C., the Hon. G. M. Weir, that this view will find favour, and if so, the inception of the Medical School, so badly needed in this Province, will probably be a fact within a reasonable time. Up-to-date, the necessity for a teaching hospital has been an almost insuperable obstacle to this realization of the hopes of those who want to see a Medical School here.

The recent election to fill the vacancies occurring yearly in the Council of the College of Physicians and Surgeons, resulted as follows: District No. 1—Dr. F. M. Bryant, of Victoria; District No. 2—Dr. G. S. Purvis, of New Westminster; District No. 3—Dr. Murray McC. Baird, of Vancouver.

Dr. H. H. Milburn is acting registrar, pending the recovery of Dr. J. A. MacLachlan from a serious illness.

The B.C. Medical Association has inaugurated a Benevolent Fund, to cover the Province as a whole. The purpose of this fund is to ensure financial assistance to doctors who are practising, or have been practising, in British Columbia, where such assistance is necessary, and to extend such aid to their wives and families, where, in the opinion of the Committee in charge, such aid is needed. The fund for this project is made up of yearly subscriptions collected with the Annual Registration Fee, from every doctor practising in B.C. A Board of Trustees has been appointed. Its members are Drs. W. E. Ainley, A. J. MacLachlan, and D. Busteed.

Senior membership in the Canadian Medical Association has been granted, on the vote of his colleagues, to Dr. R. W. Irving of Kamloops.

Dr. J. S. Kitching, who for years has done such excellent and outstanding work with the Metropolitan Health Board of Vancouver, has resigned his position. He has joined the Health Department of Hamilton, Ont.

Dr. O. O. Stromberg has left Vernon, B.C., and gone to North Battleford, Sask.

Dr. T. J. Speakman has joined the staff of the Montreal Neurological Institute. J. H. MACDERMOT

Manitoba

The Winnipeg Council has recommended that construction of an Old Folks Convalescent Hospital on the Morley Avenue site be proceeded with, in spite of advanced building costs.

A sectional meeting of the American College of Surgeons at the Royal Alexandra Hotel, Winnipeg, on April 28 and 29 attracted many surgeons and general practitioners from outside points as well as the city. Dr. Gordon S. Fahrni was chairman of the local committee of arrangements. Scientific and hospital conferences met concurrently. There was a preview of a medical motion picture, the first of a projected series of educational films, Anomalies of the Bile Ducts and Blood Vessels, Strictures of the Common Duct.

The Obstetrical and Gynæcological Section of the Winnipeg Medical Society was re-organized, after lapsing during the war years, on April 17, 1947. Dr. T. W. Dingle and Dr. P. Macdonald spoke on standardization of radium and x-ray therapy in cancer of the cervix and corpus uteri.

The Winnipeg Medical Society is to set up a Benevolent Trust Fund to be used for benevolent and educational purposes. The trustees appointed by the Society on April 18 are Drs. W. F. Tisdale, P. H. McNulty, Anna Wilson, Gordon P. Fahrni and Ross Mitchell.

The Council of the College of Physicians and Surgeons of Manitoba passed a resolution recently protesting against the proposed requirement that students applying for admission to the study of medicine be required to sign a contract agreeing to practice in Manitoba for three years after graduation.

A medical centre is developing in Winnipeg about the Medical Arts Building, at the corner of Graham and Kennedy, the first part of which was erected in 1923. Last year large additions were made to the Medical Arts Building and to the Winnipeg Clinic. The Kobrinsky Clinic occupies the second floor of a new building on Kennedy Street. The Mall Clinic building of Tyndall stone is being constructed at the corner of the Mall and St. Mary's Avenue, and work has begun on a two storey Tyndall stone and brick building at Graham Ave. and Edmonton St. Provision is made for later construction of an additional two storeys. Behind this venture are five Hollenberg medical brothers. On Edmonton Street nearby the McNulty Clinic occupies the second floor of a brick building. In addition there are four surgical supply houses within a stone's throw.

Dr. I. McLaren Thompson, Professor of Anatomy in the University of Manitoba has been elected a member of the Royal Society of Canada.

Dr. Charles Hollenberg, of Winnipeg, has recently been made a Fellow of the Royal College of Surgeons of England.

Dr. Ton L. Quong, who for several years was on the staff of King Edward and King George Hospitals, Winnipeg, has left with his wife and three children for Hong Kong where he will be a member of a clinic.

The honorary attending staff of the Winnipeg General Hospital gave a dinner on May 31 in the Macdonald room of the Fort Garry Hotel in honour of the consulting staff and of seven who had recently retired from the staff: Drs. A. Gibson, J. D. McQueen, W. E. Campbell, E. J. Washington, Gordon Chown, Oliver Waugh and A. M. Davidson. Dr. M. Rutherford MacCharles presided.

The Manitoba Sanatorium Board has agreed to take over the D.V.A. Hospital at Brandon. It will be used to care for tuberculous Polish veterans, Indians and such white patients as may be arranged for by joint agreement with the provincial government and the Board. ROSS MITCHELL

Nova Scotia

Dr. H. G. Grant, Dean of Medicine at Dalhousie University and Secretary of the Nova Scotia Branch of the Canadian Medical Association was made President of the Association of Canadian Medical Colleges at its last meeting in Montreal.

Dr. E. L. Thorne of Halifax who has been interning since his discharge from the Services has established himself in Halifax, with offices on Gottingen Street.

Dr. William Chisholm and Mrs. Chisholm have returned to their summer home at Cape George, Antigonish County, after spending the winter in Florida.

Twenty-seven students graduated in Medicine at Dalhousie University this year. This is the first class to graduate since the accelerated course adopted during the war years was abandoned. There will be a much larger group next year. Applications for first year Medicine outnumber five to one the available openings.

Dr. Robert S. Morison of the Rockefeller Foundation, New York, recently reviewed the Department of Psychiatry at Dalhousie University.

The Province of New Brunswick has recently announced a contribution of $20,000.00 to the Schools of Medicine and Dentistry at Dalhousie University.

H. L. SCAMMELL

New Brunswick

Dr. A. B. Walter, Chief of Medicine at Lancaster D.V.A. Hospital has been appointed a governor of the American College of Physicians. He succeeds Dr. H. A. Farris who has served in the same position for the past three years.

The physicians of New Brunswick are proud to congratulate Dr. A. H. Gordon of Montreal, whose election as president of the Association of American Physicians was announced in the press recently. This great Canadian was privileged to spend part of his youth in the Maritimes and has maintained many friendships here.

Dr. Darrell D. Munro of Fredericton has been granted a $1,500.00 medical fellowship by the National Research Council of Canada. Dr. Munro is a graduate of McGill in Medicine 1944. He served in the R.C.N.V.R. and on discharge he engaged in postgraduate studies under Dr. J. B. Collip, doing special research in traumatic shock. On this subject he is to present a paper at the Conference of the Federation of American Societies for Experimental Biology at Chicago.

Dr. Raymond G. Giberson of Holmesville, N.B., received the Dalhousie University Medal for high standing in each of the five years' course in Medicine at the recent Dalhousie Convocation. Dr. Giberson was also the winner of the Hon. Murray MacLaren scholarship for highest proficiency as an intern at the Saint John General Hospital for year 1946-47. A. S. KIRKLAND

Ontario

At the annual meeting of the Ontario Medical Association, the physicians of Ontario granted permission to the O.M.A. to apply for supplementary letters patent to enable them to lend money to and otherwise assist a corporation approved by the Association to provide prepaid medical or surgical care.

In the two former years when this subject was brought up, the principle of prepaid medical care was passed by Council but failed to get the support of the necessary two-thirds of the members present. But, during the past year, the Board of Directors took a plebiscite by mail. The result of this showed that the rank and file of the members, often too busy to attend an annual meeting, was in favour of the idea of prepaid medical care under the control of the medical profession. During the winter months, an active, productive, industrious committee of three studied the question. They brought in a report outlining a province-wide plan. This report was passed by Council. There was some doubt about how the voting would go and how much opposition there would be, but only 7 of the 300 or 400 voters opposed it.

An interim commission appointed by Council consisting of Dr. M. C. Watson, Toronto; Dr. John Oille, Toronto; Dr. H. D. Logan, Lindsay; Dr. A. F. Dunn, Ottawa; Dr. F. A. Brockenshire, Windsor; Dr. A. D. Pollock, Owen Sound, and Dr. M. J. Kelly, Timmins, is drawing up rules and regulations. The public reaction has been good. The public feels that the Ontario Medical Association has become actively aware of the country-wide demand for distributing the burden of medical calamity. LILLIAN A. CHASE

The Toronto East General Hospital has reorganized its staff and has now fulfilled the requirements demanded from a teaching hospital. There is as yet no agreement with the University, as the question of distance from the college laboratories is a serious one for students. The Department of Surgery is under Dr. Burns Plewes, M.A., M.B., M.S., F.R.C.S.(Edin.&C.). With Dr. L. W. Black in charge of Orthopædic Surgery. Dr. C. D. Farquharson is to be Physician in Chief, Dr. E. Cullen Bryant, D.Obst., R.C.O.G.(Lond.), head of the Department of Obstetrics and Gynæcology. Dr. J. P. Wyatt is Pathologist, Dr. A. R. McGee, Radiologist and Dr. W. E. Martin, Anæsthetist-in-Chief. Otolaryngology will be under Dr. F. Hawthorne Steele, and Ophthalmology under Dr. T. Harold Hodgson, M.A., M.D., D.O.M.S.(Eng.), Dr. A. B. James is in charge of Urology. An impressive list of assistants is also announced. The progress of this hospital from a local venture a few years ago to the status of a fully organized institution with a highly competent staff is phenomenal.

Next month we shall have a very long list of retirements from the teaching staff in the Faculty of Medicine, University of Toronto. The most prominent names in the list will be Professors Duncan Graham and W. E. Gallie. A complimentary volume of scientific essays by members of the staff in Surgery is in the press. The July issue of the *Journal* was in the printer's hands before the ceremony of presentation took place on June 19.

Dr. R. I. Harris has brought credit to himself and the University of Toronto by having been elected President of the American Association of Orthopædic Surgeons. He assumed his new duties at the Congress of the Association in Hot Springs, Va., on June 27.

The University of Toronto has inaugurated a Diploma Course in Hospital Administration. Dr. Harvey Agnew has been appointed director of the new department.

The Physiological Society of the University of Toronto was addressed on May 30 by Dr. F. J. W. Roughton, Professor of Colloid Science in Cambridge University, England. Professor Roughton's subject was "Some Recent Work on the Chemistry and Physiology of Hæmoglobin". M. H. V. CAMERON

Quebec

In the report of the last meeting of the Quebec Division of the Canadian Medical Association (June issue) omission was made of the fact that Messrs. Charles E. Frosst and Company of Montreal had contributed generously towards the program expenses of the meeting. The officers of the Division wish to acknowledge with thanks this very much appreciated assistance from Messrs. Frosst and Company.

The Principal of McGill University, Dr. F. Cyril James, has announced the establishment of a Research Institute of Bio-Physics, operating within the Faculty of Medicine. This development has been made possible by a generous grant from Messrs. Bristol-Myers Company of New York City which will cover the cost of a comprehensive five-year program of investigation in the field of medical radio-activity and cardiovascular research. This work will be under the direction of Dr. Kenneth Evelyn whose qualifications both in medicine and physics are of the highest. It will be possible with this financial aid, to assemble a team of five or six specially trained scientists to work with Dr. Evelyn. Particular attention will be directed to the study of hypertension, with the direct application of biophysical methods.

The Research Institute will consist of a clinical unit for the investigation and treatment of selected patients and a laboratory unit which will provide facilities for fundamental research, as well as for the routine analytical procedures connected with the work of the clinical unit. The laboratory unit will be housed in the Donner Building for Medical Research. The Donner Building adjoins both the Medical Building and the newly constructed Radiation Laboratories and cyclotron which is the largest in the British Empire. The Research Institute will immediately commence investigation in Medical Radio-activity and Cardiovascular research.

In announcing the grant, Mr. W. M. Bristol Jr., vice-president of Bristol-Myers Company, said: "Our Company

is enthusiastic about the project and happy to collaborate with McGill University in this field, which is one of the most important areas of Medical Research at the present time. We expect that advantage will be taken of the newer knowledge in atomic physics in an attempt to shed new light on the cause and treatment of diseases of the heart, blood vessels and kidneys.''

Dr. M. Digby Leigh, assistant professor of anæsthesia at McGill University and chief anæsthetist at the Children's Memorial Hospital, has been appointed anæsthetist in charge at the Vancouver General Hospital.

High tribute to the contributions which Dr. Leigh has made to the specialty was voiced at a farewell banquet tendered him recently at the Cercle Universitaire by the Quebec Division of the Canadian Anæsthetists' Society, under the chairmanship of Dr. Romeo Rochette, chief anæsthetist at the Hotel Dieu. Dr. Wesley Bourne, chairman of the department of anæsthesia at McGill University stated that the loss of a man of Dr. Leigh's calibre would be felt grievously here but the compensating factor was that he would make great contributions to anæsthesia in Western Canada through his energy, knowledge, techniques and skill in imparting knowledge to others.

Some 50 members of the division were present to pay tribute to Dr. Leigh who was presented with a beautiful painting by Dr. George Cousineau, on behalf of the division.

Le Dr G. H. Baril vient d'être nommé doyen de la Faculté des Sciences de l'Université de Montréal.

Le Dr Wilbrod Bonin, professeur d'histologie et d'embryologie de l'Université de Montréal vient d'être nommé ''Fellow'' de l'*American Association of Anatomists.*

L'hôpital St-Joseph de Lachine vient d'élire à la présidence de son Bureau Médical le Dr Oscar Gendron et à la présidence de son Bureau Exécutif le Dr J. Bessette.

L' ''American College of Chest Physicians'' a nommé ''Fellow'', en novembre dernier M. Herman Gauthier, médecin-chef du Sanatorium de Mont-Joli.

L'Institut Bruchési de Montréal vient d'élire à la présidence de son bureau mèdical le Dr J. C. Blais.

St-Jean Port-Joli aura l'été prochain un nouvel hôpital de 30 lits. La direction en sera confiée aux RR. SS. St-Joseph de St-Valier.

Le Dr Fernand Hébert a été élu président du Comité provincial de Défense contre la tuberculose.

Le docteur Louis-Philippe Roy, professeur agrégé de la Faculté de Médecine de Laval, chef du service d'orthopédie à l'Hôtel-Dieu et spécialiste agréé du Collège Royal des Médecins et chirurgiens du Canada, vient d'être élu membre de la Société française d'orthopédie et de traumatologie.

Lors de son récent congrès, les 15, 16 et 17 mai derniers à Québèc, l'Association Canadienne antituberculeuse s'est choisi comme nouveau président, le docteur Roland Desmeules, F.R.C.P., professeur titulaire de séméiologie, surintendant médical et chef de service à l'Hôpital Laval.

Nominations à la Faculté de Médecine, Université Laval.

Le Docteur de la Broquerie Fortier, chef des Services de Pédiatrie à l'Hôtel-Dieu et à l'Hôpital de l'Enfant-Jésus de Québec, vient d'être nommé ''Fellow'' de l'American Academy of Pediatrics, en son récent congrès à Pittsburg.

Le Docteur Pierre Jobin, professiur d'anatomie à la Faculté de Médecine, vient d'être accepté au rang des membres de l'American Association of Anatomists, lors de son récent congrès à Montréal.

Depuis quelques années déjà, nous avons vu des octrois de recherches encourager le travail des laboratoires à Laval. De récents développements, tant dans le personnel que dans le matériel, ont appelé de nouveaux et plus nombreux octrois dont il nous plaît de publier à la suite ceux de l'année en cours:

Conseil National des Recherches	$4,050.00
Subvention anonyme	2,500.00
Laboratoire Nadeau Limitée	2,000.00
Compagnie Ciba Limitée	1,000.00
Merck & Company Ltd.	1,000.00
Octroi de bourses du Conseil National des Recherches	750.00
	$11,300.00

JEAN SAUCIER

General

Brigadier C. S. Thompson, O.B.E., E.D., D.G.M.S. Canadian Army, has retired from the service and is resuming his medical practice in Montreal.

Endocrinology Fellowship at University of Montreal.—A fellowship in endocrinology, to the value of $2,500. for a period of one year, has been established at the University of Montreal under the direction of Dr. Hans Selye. Dr. Selye is a professor and director of the Institute of Experimental Medicine and Surgery at the University, a fellow of the Royal Society (Canada), and member of many American and European Associations and Societies for the Advancement of Science. Associated with Dr. Selye in this work will be Dr. Roméo de Grandpré, a graduate in medicine of the University of Montreal. The new fellowship is another in the series of grants being made regularly by Schering Corporation Limited to further explorations in recent developments in endocrinology, now recognized as a growing field of specialization.

American Board of Orthopædic Surgery.—All candidates applying for Part I of the examination of the American Board of Orthopædic Surgery after January 1, 1951 must have the following general qualifications:

(*a*) He must be a citizen of the United Sates or Canada.

(*b*) He must be a graduate of a medical school approved by the Council on Medical Education and Hospitals of the American Medical Association.

(*c*) He must have served an internship of twelve (12) months in a general hospital acceptable to the Board.

(*d*) He must have spent a year on an approved surgical residency subsequent to the completion of his internship.

The American Board of Orthopædic Surgery, Dr. Francis M. McKeever, Secretary, 1136 West 6th Street, Los Angeles 14, Calif.

The American Congress of Physical Medicine will hold its twenty-fifth annual scientific and clinical session September 2, 3, 4, 5 and 6 inclusive, at the Hotel Radisson, Minneapolis. Scientific and clinical sessions will be given the days of September 3, 4, 5 and 6. All sessions will be open to members of the medical profession in good standing with the American Medical Association. In addition to the scientific sessions, the annual instruction courses will be held September 2, 3, 4 and 5. These courses will be open to physicians and the therapists registered with the American Registry of Physical Therapy Technicians. For information concerning the convention and the instruction course, address the American Congress of Physical Medicine, 30 North Michigan Avenue, Chicago 2, Illinois.

National Cancer Institute of Canada.—The Cancer Conference reconvened in Toronto on May 12 under the chairmanship of the Honourable Paul Martin, Minister of Health for Canada. This meeting resulted in the formation of the National Cancer Institute of Canada. The interim executive of the Institute are those who signed the application for Letters Patent, namely, Dr. A. W. Blair, Dr. G. E. Richards, Mr. J. G. Stephenson, Dr. L. C. Simard, and Dr. G. D. W. Cameron. The Institute is designed to correlate and to arrange continuity in cancer research.

Among the delegates to the meeting were Professor C. H. Best, Professor William Boyd, Dr. T. C. Routley, Dr. R. D. Defries, Dr. Grant Cunningham, Dean J. A. MacFarlane and Professor H. John Ireton, all of Toronto; Profesor Lyman Duff and Professor R. P. Vivian of McGill; Dr. Louis Simard and Dr. Louis Berger of the University of Montreal; Dr. Harold Ettinger, Queen's; Dean G. E. Hall, president-elect of Western; Dr. P. A. Macdonald of Winnipeg; Dr. N. H. Gosse, Halifax; Professor J. B. Collip, chairman of the Division of Medical Research of the National Research Council; Dr. J. H. Baillie, Canadian Public Health Association; Dr. G. D. W. Cameron, Deputy Minister of Health for Canada, and Dr. J. L. Little.

The Institute is to receive a grant of $150,000 each year covering a three-year period from the King George V Silver Jubilee Cancer Fund for Canada. The permanent board of the Institute will be composed of representatives from the following organizations: two representatives of the Canadian Cancer Society; two representatives of the Medical Research Division of the National Research Council; two representatives of the Association of Canadian Medical Colleges; two representatives appointed by a committee of Directors of diagnostic and treatment cancer centres approved by the Institute; two representatives of the Royal College of Physicians and Surgeons; one representative each of the Canadian Medical Association; Canadian Public Health Association; Department of National Health and Welfare; Dominion Council of Health; and the National Federation of Canadian Universities.

Dr. J. Llew Little was appointed Registrar and Executive Secretary. He formerly served as a medical missionary to Formosa and Hong Kong, later he was Surgeon Commander in the R.C.N.V.R. before he joined the Navy, he and his wife, the former Flora Gauld, practised in Guelph, Ontario. Since his retirement from the Navy, he has been attached to the Department of National Health and Welfare, a position he relinquished in order to assume the new duties with the National Cancer Institute of Canada. LILLIAN A. CHASE

The National Gastroenterological Association takes pleasure in announcing that First Prize in its 1947 Prize Award Contest for the best unpublished contribution on Gastroenterology or an allied subject has been awarded to Dr. Frederic Duran-Jorda of Manchester, England. Dr. Duran-Jorda's paper on "Histo-Pathology of the Semi-Squamous Epithelial Layer as Found in the Colon" was selected by the judges from amongst 12 entries received from all parts of the world.

BOOK REVIEWS

Allergy in Theory and Practice. R. A. Cooke, Attending Physician and Director of the Department of Allergy, the Roosevelt Hospital, New York City. 572 pp., illust. $9.00. W. B. Saunders Company, Philadelphia and London; McAinsh & Co. Ltd., Toronto, 1947.

The subject is treated as the title of the book suggests. The sequence of topics follows a fairly conventional pattern with discussion of fundamental immunological and other scientific principles and their clinical application. Comprehensive bibliographical references are supplied. The clear literary style, and good quality of paper and printing contribute towards the pleasure derived from reading the text. Dr. Cooke's well known ability in the use of concise exact expression brings to the book a fund of studied mature opinion and comment. Up-to-date considerations of etiology and treatment of bronchial asthma, urticaria, dermatitis are well covered; and much more information is provided than one usually finds in a volume of this size, on less common but important problems such as allergies of the eye, the cardio-vascular system, digestive system, allergy of infancy and childhood; allergy to drugs, to endocrine products, physical allergy; migraine, Ménière's disease and vaso-motor rhinitis. This book is highly recommended.

Experiences with Folic Acid. T. D. Spies, Associate Professor of Medicine, University of Cincinnati School of Medicine. 110 pp., illust. $3.75. The Year Book Publishers, Inc., Chicago, 1947.

This compact, well written volume gives in readable style the author's experiences in the use of this very interesting synthetic substance in the treatment of anæmia. He reports the results obtained in the treatment of pernicious and related anæmias in 218 patients by this agent, known as folic acid, lactobacillus casei factor and pteroylglutamic acid. He states that every patient with pernicious anæmia, sprue, macrocytic anæmia of pregnancy or pellagra responded to folic acid therapy, either by mouth or parenterally. He suggests a daily dose of 20 mgm. by either route as the effective dose.

The author points out that folic acid does not always prevent neurological changes in pernicious anæmia nor does it reverse them. These observations, he states, suggest that folic acid is not the major antianæmic substance in liver extracts and yeast concentrates, both of which bring about remission in macrocytic anæmia out of all proportion to their folic acid content. It may, however, replace liver extract in the patient who is sensitive to liver. The author points out that the use of folic acid by the clinical investigator as a specific hæmatological agent will open up fresh and fertile fields of clinical research in diseases of the blood forming organs.

Biology of Tissue Cells. A. Fischer, Director of the Biological Institute, Carlsberg Foundation, Copenhagen. 348 pp., illust. $6.95. Cambridge University Press. G. E. Stechert & Company, New York; Gyldendalske Boghandel Nordisk Forlag, Copenhagen; Macmillan Co. of Canada, Toronto, 1946.

This book is a collection of experimental data obtained by means of the tissue culture techniques and arranged in relation to some of the main problems of biology. Though the author does not attempt to deal with the various problems exhaustively, he does succeed in giving an excellent presentation of countless instances in which tissue culture has furnished important contributions to the understanding of fundamental biological problems. Much of the work that is reported, some for the first time, has been carried out by the author, his associates and his students over the past twenty-five years.

As the author states, it is still too early to furnish a complete picture of the physiology of tissue cells in general, although much has been revealed by cultivating the cells outside the organism under conditions that may be altered at will. Moreover, the cultivation of pure strains of tissue cells provides the only method by which certain problems can be attacked, as, for example, the attempt to determine the exact nature of the substances required by the cells for the elaboration of new cytoplasm. Accordingly, various chapters are devoted to discussions of the interrelation of cells and medium, of senescence and rejuvenescence, of the nature of the growth-promoting substances, of the nitrogen metabolism of the cells, and of studies having to do with the respiration and glycolysis of tissue cells during actual

multiplication. Of equal importance are other chapters dealing with the peculiar nature of the tissue colonies themselves, with the fundamental problems of growth and development, and with the mechanisms of regeneration, differentiation and organization. Details of the general techniques have been omitted. There is, however, an ample bibliography.

The opinions and interpretations set forth by the author are far-reaching in their scope and vigorous in their enunciation. And while the book is recommended particularly to students and teachers of histology, embryology, physiology and biochemistry, it will be read with unusual interest by investigators in cytochemistry and cellular nutrition.

Nutritional and Vitamin Therapy in General Practice. E. S. Gordon, Associate Professor of Medicine, University of Wisconsin. 410 pp., illust., 3rd ed. $5.00. The Year Book Publishers, Chicago, 1947.

The title of the book and the fact that it is published as one of the General Practice Manuals suggest that it has been written especially for the use of the general practitioner. It is unlikely that the busy practitioner—either general or specialized—will find the text sufficiently concise or clear to be useful. In the opinion of the reviewer, the book is not well written and is difficult to read. Its style is redundant, it does not display that clarity and orderliness of presentation which are essential to good teaching and much of the space is devoted to controversial subjects. The charts, tables and organic formulæ present features which do not improve the clarity of the text. For example, two charts included in the chapter on Vitamin A (pp. 44 and 45) appear to have been misplaced; the important table of recommended dietary allowances, deserving of an explanatory chapter in itself, is inserted for no obvious reason at the end of the chapter on Vitamin A; the diagram on p. 142 representing the methyl pool is not illuminating; and some of the organic formulæ have been set up in such a way that they will appear to the clinical reader to be a bewildering mass of symbols. The aim of providing the general practitioner with a concise manual of the scientific basis of nutritional therapy is a worthy one but the reviewer does not believe that, with this text, the author has accomplished his purpose.

Parenteral Alimentation in Surgery. R. Elman, Associate Professor of Clinical Surgery, Washington University School of Medicine, St. Louis, Mo. 284 pp., illust. $4.50. Paul B. Hoeber, Inc., New York, 1947.

This is a very timely review of the whole subject of parenteral feeding. While it is written by a surgeon for surgeons, the information should be of equal value to doctors in all branches of clinical medicine since the problem of tissue requirements remains fundamentally the same.

The writer attacks the subject in a broad way which never allows the reader to become lost in a maze of intricate details. After an appropriate and interesting historical background, the six nutritional requirements—*i.e.*, water, electrolyte, carbohydrate, protein, fat and vitamins—are discussed in a clear and orderly fashion. An example of parenteral fluids (blood and plasma) in the treatment of shock, which is regarded as nutrition that is needed in a hurry, in contrast to the less urgent but similar requirements of starvation. Throughout the book there is a constant flow of proof and logic which will satisfy even the most doubting. After establishing the definite amounts of the nutritional requirements in health and under abnormal conditions, the author then describes the methods of supplying these amounts parenterally. In this way the whole subject of parenteral nutrition is brought up to date.

One will rarely find a technical book that is so clear, so interesting and of such practical importance.

Pædiatric Gynæcology. G. C. Schauffler, Assistant Clinical Professor of Obstetrics and Gynæcology, University of Oregon Medical School. 380 pp., illust., 2nd ed. $6.00. The Year Book Publishers, Inc., Chicago, 1947.

This book reviews the normal anatomy, physiology and developmental processes of the female genital tract. The volume is written to be clinically helpful rather than scientifically exhaustive. Congenital abnormalities which occur in the female are well described. This is in reference to the type of abnormalities and their associated conditions. Treatment is also described.

Methods of diagnosis relative to vaginal and pelvic infections, culture and spread techniques are mentioned. Disorders during adolescence and those relative to the onset of menstruation are also described. These include discussions relative to the difficult problems of masturbation, enuresis and childhood sex pattern deviations.

The chapters on special urologic conditions and lower bowel lesions should interest those who are in active clinical practice. This is also the case relative to basic standards of hormone units and the available commercial hormone preparations.

This book should prove useful to those who are particularly interested in gynæcological or pædiatric practice. In addition, it will serve as a reference text to domestic court jurists, medico-legal consultants and social service workers.

Postgraduate Obstetrics. W. F. Mengert, Professor and Chairman, Department of Obstetrics and Gynæcology, Southwestern Medical College. 392 pp., illust. $5.00. Paul B. Hoeber, Inc., New York, 1947.

This is a practical treatise on the management of normal obstetrics and the commonly encountered abnormalities of pregnancy, labour and the puerperium. Needless detail is avoided, theoretical considerations receive scant attention and rare and unimportant conditions are omitted or only briefly mentioned. The book is divided into three sections: pregnancy, labour and the puerperium, with an appendix on nursing technique. Subject matter is presented with clarity and simplicity. If the book has any fault it is one of over-simplification. The chapters on difficult and delayed labour, and the toxæmias of pregnancy, are typical examples of the manner in which complex and controversial problems are discussed. These problems are concisely and clearly presented and their management outlined in an orderly and systematic manner. The book is filled with good, sound, conservative advice for the management of the pregnant patient throughout pregnancy, labour and the puerperium. Practitioners, whether with scant or extensive experience in obstetrics, will find this book most valuable.

Practical Anæsthetics. H. Parry-Price, Anæsthetic Specialist to the Royal Navy. 127 pp., illust. $3.00. John Wright & Sons, Bristol; Macmillan Co. of Canada, Toronto, 1946.

The author does not intend this book to take the place of a reference or textbook, but he has covered briefly the techniques of general, spinal, intravenous, rectal and local anæsthesia. In his limited space he has been unable to cover the techniques used with the different anæsthetic agents in spinal anæsthesia, so in his chapter on this subject he describes mainly the use of the 1 to 1,500 solution hypobaric nupercaine. In the description of gas machines he has made use effectively of some fine illustrations. He describes several types of apparatus, but has omitted the Foregger and Heidbrink gas machines which are used extensively in Canada and the United States.

The author has, however, covered all the fundamentals and has made his book interesting by telling the difficulties and problems which he has met with in his twenty-five years' experience in anæsthesia, and which every anæsthetist is bound to meet. His ex-

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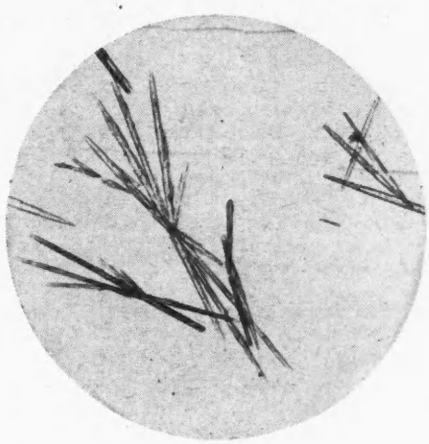

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perience in the Service during the last year has qualified him to discuss the difference between service and civilian anæsthesia.

This book can be highly recommended to all those interested in anæsthesia.

Practical Physiological Chemistry. P. B. Hawk, President, Food Research Laboratories, Inc., New York; B. L. Oser, Director, Food Research Laboratories, Inc., New York; and W. H. Summerson, Associate Professor of Biochemistry, Cornell University Medical College, New York. 1,323 pp., illust., 12th ed. $11.00. The Blakiston Co., Philadelphia and Toronto, 1947.

The twelfth edition of this standard text will be welcomed by all who know it. Many changes in the field of physiological chemistry have occurred in the ten years since the last edition was issued and these are well covered here without the deletion of any fundamental principles. The chapter on the vitamins is complete and well documented, both as to clinical application and practical methods. New material on hormones and the use of isotopes in the study of metabolism are thoroughly presented, as is the principle and use of the Warburg slice technique. This is one of the most valuable complete reference books on this subject, covering as it does both theoretical and practical aspects.

Psychiatric Interviews with Children. Edited by Helen Leland Witmer. 443 pp. $4.50. The Commonwealth Fund, New York, 1946.

This publication represents a distinct contribution to the field of child psychiatry. The book is an excellent training manual for students and practitioners in child guidance. After three introductory chapters concerning the dynamics of child guidance and therapy, the book is devoted to a detailed recording of interviews with child patients with a variety of behaviour problems. Six of the cases fall in the age range of seven to nine, three are adolescents and one a preschool child.

The point of view is adopted that therapy with children only can be successful with concurrent case work service to parents. The social worker's rôle in helping to solve their emotional conflicts, particularly as related to attitudes to their children, is stressed. This book, however, is more concerned with the techniques the child therapist uses with his child patient; only progress summaries of case work with parents are given indicating, among other things, the plans made with the social worker to introduce the child to the clinic and insure acceptance on the part of the child of help from the therapist. The social worker also provides the therapist with history data and the course of the child's adjustment to the home during therapy.

Radiology for Medical Students. F. J. Hodges, Professor and Chairman, Department of Roentgenology, University of Michigan; I. Lampe, Associate Professor, Department of Roentgenology, University of Michigan; J. F. Holt, Assistant Professor, Department of Roentgenology, University of Michigan. 424 pp., illust. $6.75. The Year Book Publishers, Inc., Chicago, 1947.

This book is relatively short in distinction from many textbooks which by their mere size tend to discourage and bewilder the student. The Diagnostic Section outlines in a clear, concise manner the general principles of radiological diagnosis of the Head, Spine and Extremities, Thorax, Gastro-intestinal Tract and Genito-urinary Tract. Many excellent illustrations are included, although no attempt is made to describe or illustrate all the rare conditions which may be encountered. The Therapeutic Section is exceptionally well written and commences with a general view of therapeutic radiology which is clearer and more comprehensive than many similar sections in other much larger books designed for postgraduate students. This is followed by a discussion of radiation therapy of various organs and diseases either as a sole method of treatment or in combination with other forms of therapy, particularly surgery. No attempt is made to overemphasize the importance of radiation therapy and it is frankly admitted that in many lesions the result of such therapy is poor or negligible.

Although many portions may be read with advantage by the graduate student its chief interest is for the undergraduate. Combined with an adequate course of lectures it should prove invaluable and it is hoped that it will soon be adopted as a text in many of our medical schools.

Synopsis of Orthopædic Surgery. A. D. LeVay, Honorary Orthopædic Surgeon, Woolwich Memorial Hospital. 242 pp., illust. 15s. H. K. Lewis & Co. Ltd., London, 1947.

This small volume condenses into 228 pages the common orthopædic disorders and fulfills the author's intention of being a synopsis of the subject. The first part of the book deals with the disease processes and concerns itself with systemic and local diseases of the skeleton and of the joints. This is followed by the exposition by regions and concludes with a chapter on neurological disorders. The interpretation of orthopædics throughout is in the orthodox manner and traumatic surgery including fractures and dislocations is excluded. No detailed description of operative procedures could of course be included in a work of this size.

This book will find a place for senior students and for revision for candidates for higher examinations.

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Philosophy and Medicine in Ancient Greece. W. H. S. Jones, Litt.D., F.B.A. Supplements to the Bulletin of the History of Medicine, Edited by H. E. Sigerist. No. 8. 100 pp. $2.00. The Johns Hopkins Press, Baltimore, 1946.

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Anatomy and Physiology. C. F. V. Smout, Assistant Professor of Anatomy, University of Birmingham; and R. J. S. McDowall, Professor of Physiology, University of London, King's College. 470 pp., illust., 2nd ed. $6.75. Edward Arnold & Co., London; Macmillan Co. of Canada, Toronto, 1947.

1946 Year Book of General Therapeutics. Edited by O. W. Bethea, Professor of Clinical Medicine, Tulane University School of Medicine (retired). 443 pp., illust. $3.75. The Year Book Publishers Inc., Chicago, 1947.

Future for Preventive Medicine. E. J. Stieglitz, M.D., F.A.C.P. 77 pp. $1.00. The Commonwealth Fund, New York, 1945.